MEDICAL PROCEEDINGS

MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

P.O. Box 1010 · Johannesburg Posbus 1010 · Johannesburg

Vol. 4 31 Mei 1958 May 31 No. 11

REDAKSIONEEL · EDITORIAL

'N LANKSPEEL-OPNAME VAN HART-GELUIDE EN -GERUISE*

Die ontleding van hartgeruise kan aansienlike moeilikheid oplewer. Vir die student wat in die beginsels van auskultering ingewy word, is dié ondervinding dikwels 'n beproewing. 'n Gedeelte van die moeilikheid verbonde aan die beluistering van die hart moet toegeskryf word aan die feit dat die student en die leermeester selde in staat is om gelyktydig na 'n hartgeruis te luister—iets wat gelyktydige kommentaar op en 'n bespreking van die abnormale patroon van geluide moontlik sou maak.

Die pogings wat aangewend is om hartgeruise aan 'n hele klas uit te saai, het met soveel verdraaiing gepaard gegaan dat hierdie praktyk nooit eintlik goed gevestig geraak het nie.

Die opname van hartgeluide en -geruise op hierdie lankspeelplaat is 'n merkwaardige prestasie, veral weens die aansienlike getrouheid waarmee abnormale hartgeluide gereproduseer word.

Die kommentaar (deur dr. D. G. Geckeler wat hom deurgaans streng bepaal by die benamings wat deur die Amerikaanse Hartvereniging aanvaar is) is ingeskakel by 'n ontleding van iedere opeenvolgende reeks. Die auskulteringsbevindings is met 'n konven-

* Die nommer van hierdie plaat is BLD 7089 (CBS Masterworks).

A LONG-PLAYING RECORD OF HEART SOUNDS AND MURMURS*

The analysis of heart murmurs may be a matter of considerable difficulty. For the student being inducted into the principles of auscultation, the experience is often a trial and a tribulation. Part of the handicap in learning about heart murmurs is due to the fact that the student and the teacher are seldom able to listen to a heart murmur simultaneously—a practice which would permit comment and discussion of the abnormal pattern of sounds 'on the wing'. Attempts to broadcast heart murmurs to a whole class have been associated with so much distortion that the practice has never become established.

The recording of heart sounds and murmurs on this long-playing disc is a remarkable achievement, particularly because of the considerable verisimilitude with which the abnormal heart sounds have been reproduced.

The commentary (by Dr. D. G. Geckeler, who adheres rigidly to the nomenclature adopted by the American Heart Association)—is geared to an analysis of each sequence. Auscultatory findings were recorded on a conventional tape recorder. This allowed illustrations to be made up at slow heart rates without altering the heart sounds or the murmur being studied.

*This record is identified as BLD 7089 (CBS Masterworks).

sionele bandopnemer opgeneem. Dit het dit moontlik gemaak om toeligtings tydens stadige harttempo's te verstrek sonder enige verandering van die hartgeluide of -geruise wat bestudeer word.

‚Dit is gedoen deur die eerste geluid, tweede geluid, en geruis of geruise te identifiseer, en reekse hartsiklusse dan op te sny en eerste geluide, tweede geluide en die geruis of geruise weer met mekaar te verbind, met skoon band tussen in. Teen hierdie stadige tempo's is die student in staat om die deel van die hartsiklus waar die geruis voorkom, te vind, en om die verskillende kenmerke daarvan, dit wil sê die toonhoogte, die duur, of dit blasend, rof of musikaal is, en die tipe—crescendo of decrescendo —beter te begryp. Hierdie selfde metode het dit ook moontlik gemaak om 'n geruis as sodanig van die hartgeluide te isoleer, om dit te hergroepeer en om dit uiteindelik teen die oorspronklike tempo terug te speel.'

Die hartgeluide en -geruise kan op 'n baie bevredigende en doeltreffende wyse gehoor word met behulp van 'n gewone draadloosstel van die standaard-tipe. Addisionele elegantheid sal waarskynlik aan die reproduksie verleen kan word met 'n ‚high fidelity'-toestel, hoewel dit skaars noodsaaklik is.

Die demonstrasie van die verskeidenheid van mytergeruise is veral suksesvol en insiggewend. Dit geld ook vir die voorbeelde van die derde hartgeluid, ontydige kloppings, koppeling, fibrillasie, galoppering, ens. Die hartsak-friksievrywing is ongetwyfeld moeilik om te hoor. Dit word erken deur die kommentator wat aandui dat 'n artefak in die opname ontwikkel het. Die toeligtings sluit onder meer in geruise wat oor die verskillende auskulteringsoppervlaktes gehoor is, voorbeelde van die verwarring wat deur asemhalingsgeluide veroorsaak kan word, ens. Daar is ook 'n voortreflike demonstrasie van die geruis van 'n duidelike ductus en die geruis afkomstig van 'n arterio-veneuse fistel.

Die tegniek om hartgeluide en -geruise te ontleed deur middel van 'n lankspeelplaat is geskik vir klein groepe, liewer as groot klasse. Hierdie lankspeelplaat maak dit ook moontlik om 'n besondere reeks oor en oor te speel so dikwels as wat deur die bespreking en debat vereis word. Dr. Geckeler doen 'n beroep op die luisteraar om homself op te lei om na net een ding op 'n keer te luister—'n reël wat, as dit stiptelik nagekom word, die begrip van 'n abnormale hartpatroon heelwat kan vergemaklik.

Die beskikbaarstelling van hartopnames op lankspeelplate behoort die lot van studente sowel as hul leermeesters aansienlik makliker te maak. Met behulp van 'n enkele plaat is dit tans moontlik om 'n ware museum van hartpatologie te bestudeer. Die student, of hy nou al 'n ongegradueerde dan wel 'n nagegradueerde is, is nie langer afhanklik van die kliniese materiaal wat op enige besondere dag in 'n hospitaalsaal beskikbaar is nie. Maande van saalwerk kan nou saamgevat word in 'n halfuur van intelligente besluistering en ontleding.

Dr. Geckeler het 'n waardevolle pioniersbydrae tot die bestudering van auskulteringsbevindings gelewer wat met dankbaarheid deur studente sowel as praktisyns erken behoort te word.

'This was done by identifying the first sound, second sound, and murmur or murmurs and then cutting up series of cardiac cycles and splicing together, with blank tape between first sounds, second sounds, and the murmur or murmurs. At these slow rates the student is able to locate that part of the cardiac cycle where the murmur occurs and to understand better its different features, that is, its pitch, its length, whether it is blowing, rough, or musical, and crescendo or decrescendo in type. And by this same method a murmur itself can be isolated from the heart sounds, then regrouped, and finally replayed at its original rate.'

The heart sounds and murmurs can be heard very satisfactorily and adequately on a standard domestic type of wireless set. No doubt very great additional elegance can be achieved with high-fidelity reproductions, although this hardly seems essential for the purpose.

The demonstration of the variety of mitral murmurs is particularly successful and instructive as also are the examples of the third heart sound, premature beats, coupling, fibrillation, gallop, etc. The pericardial friction rub is undoubtedly difficult to hear. This is recognized by the commentator who indicates that an artifact developed in the recording. The illustrations include, *inter alia,* murmurs heard over the various auscultatory areas, examples of the confusion which can be introduced by breath sounds, etc. There is an excellent demonstration of the murmur of patent ductus and the bruit from an arterio-venous fistula.

The technique of analysing heart sounds and murmurs by means of a long-playing record is suitable for small groups rather than large classes. The LP record also permits a particular sequence to be re-played as often as discussion and debate require. Dr. Geckeler urges the listener to train himself to listen for only one thing at a time—a principle which, if closely attended to, will facilitate the understanding of abnormal heart patterns.

The introduction of heart recordings on long-playing records should make much easier the lot of the student as well as the teacher. On one record it is now possible to study a veritable museum of cardiac pathology. The student (whether undergraduate or post-graduate) is no longer dependent on the clinical material which happens to be in the wards on a particular day. He can compress months of ward-rounds into half an hour of intelligent listening and analysis.

Dr. Geckeler's pioneering contribution to the study of auscultatory findings is very considerable and should find appreciative recognition from students as well as practitioners.

ACUTE POLIOMYELITIS*

A STUDY OF THE CLINICAL MANIFESTATIONS OF FIFTY CASES SEEN AT THE CHILDREN'S HOSPITAL, JOHANNESBURG, DURING THE 1948 EPIDEMIC WITH SPECIAL REFERENCE TO THE MANAGEMENT IN THE ACUTE PHASE

J. L. BRAUDO, M.B., B.CH., M.R.C.P. (EDIN.)
Johannesburg

(*Continued from p.* 237)

III. CLINICAL FEATURES: AGE, SEX, SYMPTOMS AND SIGNS OF 50 CASES OF ACUTE POLIOMYELITIS

Hypertension. Blood pressure estimations were recorded in every case and repeated on at least 3 separate occasions. Hypertension was diagnosed when both the systolic and diastolic pressure exceeded the normal range by at least 10 mm. Hg. The latter criteria were accepted in the 1946 Minnesota epidemic.[29] In this series hypertension was recorded in 16 cases (Fig. 3). The severest degree was seen in the cases that died, 2 of which in addition showed small pulse pressures, 15 and 20 mm. Hg. respectively. The distribution of the hypertension was as follows: 6 cases in the bulbar and bulbo-spinal group, 6 cases in the

* The References will be published at the end of the concluding article in this series.

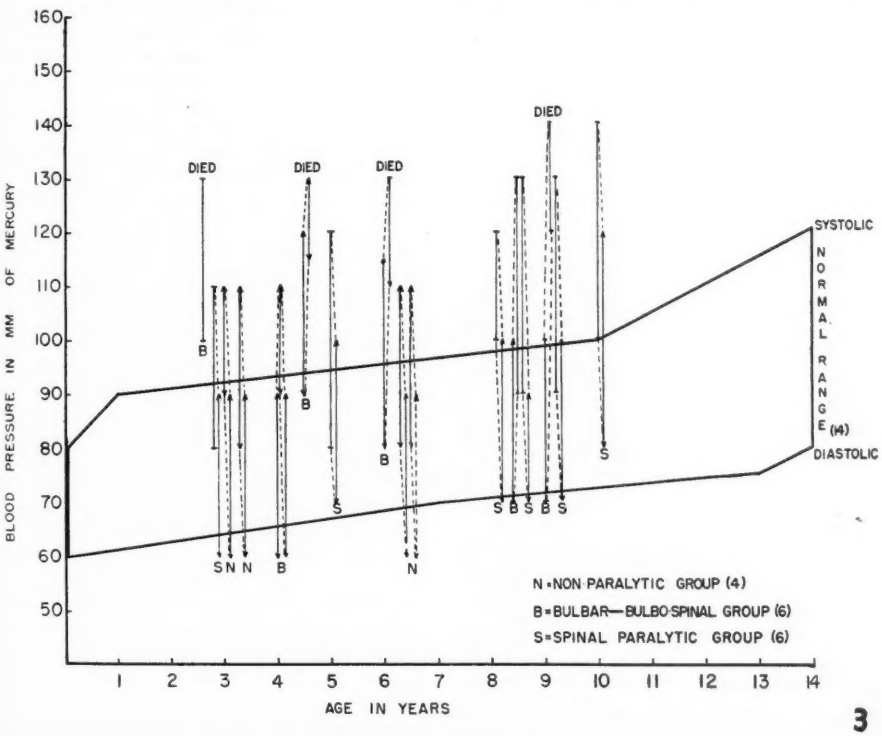


Fig. 3. Hypertension in 50 cases of poliomyelitis. [*After* Grulee, Jr, C. G. and Panos, T. C. (1948): *Epidemic Poliomyelitis*, Amer. J. Dis. Child., **75**, 24].

spinal paralytic group and 4 cases in the non-paralytic group. Thus hypertension was not confined to the bulbar variety of the disease. Grulee and Panos[29] noted the greatest incidence in the bulbar variety of the disease, which included those cases with spinal involvement as well. A comparison of the incidence and duration of this symptom in the 2 series is shown in Table 8.

TABLE 8: HYPERTENSION IN THIS SERIES COMPARED WITH THAT IN 1946 MINNESOTA EPIDEMIC[61]

Epidemic	*Bulbar*	*Incidence of Hypertension*	*Duration of Hypertension*
Minnesota[61] ..	70 cases	51 cases	Average 3·4 days
This series ..	14 cases	6 cases	Average 4·3 days
	Spinal Paralytic		
Minnesota[61] ..	41 cases	18 cases	Not stated
This series ..	19 cases	6 cases	Average 4·3 days
	Non-Paralytic		
Minnesota[61] ..	36 cases	14 cases	Not stated
This series ..	17 cases	4 cases	Average 6·3 days

Although the series described in this report is small, the figures of the incidence of hypertension in the non-paralytic and spinal paralytic groups simulated each other, whereas the major differences appeared in the consideration of the 2 bulbar groups. This could well be due to the greater frequency of the paralysis of pharyngeal musculature giving rise to anoxia of the vasomotor centres in the Minnesota epidemic. The duration of the hypertension was very similar in both epidemics. A raised blood pressure was an unusual finding in pyrexial diseases and its presence in 16 of 50 cases in this series and in 83 of 177 cases recorded by Grulee and Panos[29] was significant.

Hypertension was not commented upon in the epidemics described by earlier authors,[30, 33] but has been the subject of numerous articles in recent years. Weinstein and Shelokov[39] noted hypertension in 7% of 428 patients. Eight cases occurred in children under 16 years of age, 5 of whom had bulbo-spinal poliomyelitis, 2 spinal poliomyelitis and one bulbo-encephalitic poliomyelitis.

These authors found a constant association between cyanosis and a raised blood pressure. The disappearance of the hypertension was hastened when respiration was made more efficient, thereby improving oxygenation of the tissues. This was the opinion of Grulee and Panos[29] as well, but it did not explain those cases in which hypertension was present without clinical anoxia.

Platou[40] considered fluctuation in blood pressure or a definite rise to be one of the most valuable signs predicting the onset of bulbar involvement.

McDowell and Plum[41] noted hypertension in 45 of 95 adult patients. The highest incidence occurred in patients suffering from bulbo-spinal palsy or paralysis of all 4 limbs. These were the most severely ill, most of whom required artificial respiration. The hypertension was more severe and of longer duration in patients needing artificial respiration. In those respirator cases surviving, the average duration of the hypertension reached 98 days at the time of the report. In the hypertensive non-respirator group the blood pressure remained elevated for an average of 6 days. Of the 10 patients whose blood pressure remained elevated 3–12 months after the onset of their illness, all needed artificial respiration for at least 2 weeks in the earlier part of their illness. The part played by anoxia or high blood CO_2 concentration in the production of hypertension in these cases is difficult to assess.

Frequent CO_2 combining power and arterial oxygen saturation studies failed to reveal hypercapnia or anoxaemia in many of the patients. Brief periods of hypercapnia or anoxaemia may occur undetected and conceivably intensify the already present hypertension. Many patients develop hypertension hours before any significant reduction in their ventilatory capacity, as determined by spirometry, is noted. Furthermore, brief periods of anoxia or hypercapnia would not be expected to produce lasting hypertension. The explanation probably lies in the pathology of poliomyelitis. Both the hypothalamus and medullary reticular substance contain autonomic nuclei which produce hypertension when stimulated.[42] Bodian[43] has demonstrated damage to brain-stem autonomic structures in many patients suffering from poliomyelitis regardless of the extent or severity of the clinical signs of the disease. Vickers[44] found hypertension in 11 of 44 young adults who had had poliomyelitis many years before and suggested that the non-respiratory group

carried an increased susceptibility to the future development of hypertension.

Hypertension as part of an alarm reaction or apprehensive state is well known. It has been produced experimentally in such circumstances[45] and was noted transiently in situations endangering human security, such as the Texas City disaster of 1947,[45] and in soldiers after long periods of combat duty.[47]

The mere presence of apprehension in many cases of poliomyelitis without respiratory involvement, and its increase in those cases showing respiratory or bulbar involvement, could elevate the blood pressure. The fact that the blood pressure was elevated in many young patients before the paralysis or difficulty in respiration appeared, suggested the involvement of the medullary autonomic centres in these instances. Anoxaemia, raised CO_2 combining power of the blood and the circulatory effects resulting from the use of the respirator, were all contributing factors, not primary causes. The renal hypertensive mechanism could be stimulated by renal anoxia as part of the generalized anoxic state, or as part of the vaso-spastic state affecting the renal arteries. Significant titres of vaso-excitor material were found in 5 patients tested by Lee in Shorr's laboratory.[48] The titres were very high in 3 cases with hypertension who received artificial respiration. Vaso-excitor material was present, but lower, in 2 non-hypertensive cases.

Vasomotor phenomena, muscle tenderness and skin hyperaesthesia. The features included under vasomotor phenomena comprise coldness, hyperhidrosis and pallor of the extremities, especially the feet. Although no patients complained of these disabilities before admission, they were present in 20 cases. Ten cases belonged to the spinal paralytic, 8 to the non-paralytic and 2 to the bulbo-spinal group. The vasomotor changes were associated with muscle tenderness and skin hyperaesthesia in every case and all the features persisted for 3–6 days. The commonest muscle groups demonstrating this sensitivity to pressure were those of the calves, thighs, spine, shoulders and elbows. The slightest stroking of the skin, especially of the limbs, of these patients produced great discomfort.

The earlier writers, Peabody *et al.*[30] and Ruhräh and Mayer[33] noted a peculiar blanching and pallor of the skin in various areas, but did not especially comment on temperature. These features were most often seen in the meningeal form of the disease. Most of the modern literature, however, is confined to the interference with the circulation of the legs and feet during the chronic phase of poliomyelitis.[49, 50] The symptomatology included cyanosis on dependency or in the cold, hyperhidrosis, trophic changes in the skin and nails, swelling of the feet and frequently hyperaesthesia.[49] The vascular change was not directly related to the degree of paralysis of an extremity and its appearance in the non-paralytic group was also noted by Stenport,[50] who mentioned the presence of vaso-constriction in the acute stage in some patients which persisted into the chronic stage. In one case in this series the peripheral circulation was disturbed during the acute phase of the disease, disappeared during the period of hospitalization and recurred when the child went home. The most striking difference between this series and those referred to was the frequency with which vasomotor disturbances appeared during the acute phase of the disease.

Pharyngeal injection was noted in 23 cases, 11 of which belonged to the non-paralytic, 6 to the spinal paralytic, 3 to the bulbo-spinal and 3 to the bulbar group. Redness of the pharynx was seen in 248 of 450 cases (56%) described by Grulee and Panos,[29] but was not a prominent feature in the older epidemics.

Hoyne's sign, a peculiar weakness of the flexor muscles of the neck, was present in 15 cases; 9 in the spinal paralytic, 3 in the non-paralytic group and 3 in the bulbo-spinal group. It was always associated with severe neck stiffness and usually persisted for as long as the latter disability. In many of the cases it seemed that the patient just did not want to hold his head up because it stretched the stiff, painful, posterior neck muscles and aggravated the existing discomfort. This sign was also recorded by Peabody.[30]

Flushed facies with circumoral pallor were noted in 12 cases; 6 in the spinal paralytic, 4 in the non-paralytic and 2 in the bulbo-spinal group. It usually disappeared when the temperature returned to normal.

Nystagmus was present in 3 fatal cases. It was horizontal in all the cases, horizontal and vertical in 2 cases and vertical and rotatory in one case. The author saw several cases of true horizontal nystagmus in all 4 varieties of poliomyelitis during the 1947–8 Johannesburg epidemic and its appearance in only fatal cases in this series was probably coincidental. Transitory horizontal nystagmus was recorded in 17 of the 500 cases analysed and occurred in 7 cases of the non-paralytic, 4 of bulbar and 6 of the bulbo-spinal group. However, difficulty in fixing the eyes on a specific object was encountered to a greater or less degree in

all the cases exhibiting apprehension, many of whom showed irregular jerking movements of the eyes but no true nystagmus. Ruhräh and Mayer[33] reported nystagmus accompanying paralysis of certain cranial nerves in cases with ataxia and independently of cranial nerve palsy or ataxia. The condition was usually transitory.

Pupillary disturbances are rarely seen without cranial nerve involvement. Hippus was observed in 3 fatal cases in this series, all of whom displayed other signs of polioencephalitis. This sign is a well-known feature of the disease entity 'encephalitis'. One case with a third nerve palsy showed in addition narrowing of the palpebral fissure and a small pupil on the affected side with absence of sweating over that side of the face—a typical Horner's syndrome. The palpebral fissure narrowing and small pupil are recorded by other observers.[4]

TABLE 9: SYMPTOMS IN 50 CASES OF ACUTE POLIOMYELITIS

Clinical Features	*Non-Paralytic Form* (17 *Cases*)	*Spinal Paralytic Form* (19 *Cases*)	*Bulbo-Spinal Form* (7 *Cases*)	*Bulbar Form* (7 *Cases*)
Prodromata				
Diphasic	3	7	4	1
Straggling	7	5	2	2
Invasive	7	7	1	4
Concomitant illness in family	2	2	2	2
Operations, Injections and Fatigue	Tonsillectomy Boxing	1 Trauma 2 Injections	Tonsillectomy	Swimming
Symptoms				
Fever ..	11	15	4	5
Spinal stiffness and pain ..	5	10	3	0
Headache ..	5	7	3	1
Weakness ..	0	9	2	7
Limb pain	2	7	1	4
Vomiting ..	4	0	0	0
Restlessness	3	0	0	1
Drowsiness	2	0	0	0
Urinary Difficulty	1	2	0	0
Convulsions	2	1	1	0
Abdominal pain ..	0	1	1	0
Abnormal Gait ..	1	2	0	0
Sore-throat	0	2	0	0
Diarrhoea	1	0	0	0
Sweating ..	1	0	0	0

Tachypnoea was observed in 3 cases. Irregularity of respiratory rhythm and increased respiratory rate were very obvious in one case that recovered and all 4 that died.

Coma was present in 2 cases. One patient died while the other remained comatose for 3 days. On recovering consciousness quadriplegia was noted.

Insomnia was a prominent feature in 2 cases both of which died.

Retention of urine persisted for 2 days in one case belonging to the spinal paralytic variety. It was associated with haematuria—cause unknown.

The salient symptoms and signs are summarized in Tables 9 and 10.

Of all the clinical varieties of poliomyelitis the non-paralytic one is the least important,

TABLE 10: SIGNS IN 50 CASES OF ACUTE POLIOMYELITIS

Clinical Features	*Non-Paralytic Form* (17 *Cases*)	*Spinal Paralytic Form* (19 *Cases*)	*Bulbo-Spinal Form* (7 *Cases*)	*Bulbar Form* (7 *Cases*)
Signs				
Tachycardia	15	17	7	7
Pyrexia ..	13	17	7	7
Muscle stiffness ..	15	16	4	4
Apprehension and irritability ..	9	16	3	7
Muscle weakness ..	0	17	7	2
Constipation	14	16	3	5
Pharyngeal infection ..	11	6	3	3
Tremor ..	6	13	3	3
Hyperreflexia ..	13	12	1	3
Hyporeflexia and areflexia	1	11	4	0
Vasomotor phenomena	8	10	2	0
Muscle tenderness	8	10	2	0
Skin hyperaesthesia ..	8	10	2	0
Hoyne's sign	3	9	3	0
Hypertension	4	6	3	3
Flushed face	4	6	2	0
Nystagmus	0	0	2	0
Hippus ..	0	0	3	0
Retention of urine ..	0	1	0	0
Delirium ..	0	0	1	0
Insomnia ..	0	0	2	0
Irregular pulse rhythm ..	0	0	2	0
Coma ..	0	0	2	0
Tachypnoea	0	0	3	0

since it is not responsible for death and little if any morbidity. The pure bulbar paralytic group excluding involvement of the respiratory and vasomotor centres, carries an insignificant mortality if correct therapy is instituted. On the other hand, the bulbo-spinal and spinal paralytic forms are the most serious, since the vital respiratory and vasomotor centres as well as the important peripheral breathing musculature are involved. It would, therefore, be of interest to discuss the clinical features of the various groups individually. Brown and Baker[37] divided the bulbar and bulbo-spinal varieties into 5 groups:

i. Cranial nerve nuclei.
ii. Respiratory centre.
iii. Circulatory centre.
iv. Encephalitic.
v. Bulbar lesions combined with paralysis of peripheral respiratory musculature.

These divisions are arbitrary since very few cases belong to one group alone, e.g. encephalitic manifestations may be present in all varieties of the disease. Nevertheless, the divisions are important clinically since they guide treatment and influence prognosis. In this series, examples of all these groups except group 3 are found.

i: *Bulbar or Cranial Nerve Nuclei Group* (8 *Cases*). The presenting symptom in this group was paralysis of the musculature supplied by the specific cranial nerve. Most of the patients complained of fever, 4 vomited and 2 had neck discomfort, but there was no instance of back discomfort. An analysis of the signs in this group showed the following features:

The incidence of pyrexia, tachycardia and constipation was high, but only 4 cases exhibited neck and spine stiffness, 3 showed hyper-reflexia and tremor of the outstretched hands and 2 apprehension, whereas irritability and vasomotor phenomena together with muscle tenderness and skin hyperaesthesia were not recorded. Hypertension, on the other hand, was present in 3 cases. The features regarded as encephalitic were not conspicuous in this group.

ii: *Respiratory Centre Group.* There were no pure cases of this group in this series, but 3 cases showed the characteristic clinical features associated with involvement of the respiratory centre. One case was combined with vocal cord paralysis, whereas the other 2 had limb and respiratory weakness as well. Symptoms of respiratory centre involvement usually occurred 2–3 days later than those due to the cranial nerve nuclei. The features described by Brown and Baker,[37] viz. irregularity of rhythm and respiration with a tendency to shallowness and prolonged intervals between inspiration, passing into periods of apnoea and finally cessation of respiration, were also noted in these cases. At the same time the pulse rate and temperature rose with a rise or fall in blood pressure and the patient eventually became cyanosed. Irregularity and shallowness of respiration were seen in 2 instances despite an adequate airway and continuous oxygen administration. Two of the fatal cases demonstrated a shock-like form of respiration in which there was a sudden vigorous inspiratory effort with no apparent expiratory component at all. In one case the rhythm was regular, in the other it was irregular, but both had rapid respiratory rates.

iii: *Circulatory Centre Group.* Brown and Baker[37] observed only a few patients in whom circulatory collapse was the outstanding symptom. They ascribed certain clinical features due to failure of the central control of the circulation. The patients have a dusky red, flushed, florid appearance. The lips are a deep cherry red colour. The pulse is very rapid, often irregular and sometimes difficult to palpate. The pulse pressure is small, even though the systolic pressure is at a normal level. The blood pressure has a downward trend and becomes unobtainable. Terminally, the patient goes into shock with lividity, a cold and clammy skin and hyperthermia. Only some of these features were present in 2 fatal cases in this series. These patients exhibited extreme tachycardia and irregular rhythm, but maintained elevated blood pressures until their demise. One case exhibited a small pulse pressure and hyperthermia but circulatory collapse was not seen in this series.

iv: *Encephalitic Group.* Encephalitic symptoms were well described by Wickman,[4] who stressed the profound comatose state of some patients. Other authors[19, 30] were impressed with the extreme restlessness and insomnia as well as the anxiety and apprehension in the rapidly progressive cases. Varying degrees of stupor and coma from which the patients could be easily aroused and lasting 3–16 days were seen by Peabody *et al.*[30] Grulee and Panos[29] diagnosed encephalitis in 78 (17%) of their entire reported series. The cerebral encephalitic was characterized by irrationality, personality disturbances, hyperexcitability, confusion, extreme listlessness, excessive irritability, somnolence and diplopia. Baker[51] reported encephalitic symptoms in 133 (72%) of his series. They were characterized by apprehension, irritability, restlessness, twitch-

ing and jerking of the facial muscles and to a lesser degree of the extremities, flushed face, tremor of the outstretched hands, insomnia and extremely rapid movements. In this series of 50 cases apprehension, anxiety, hyper-reflexia, tremor of the outstretched hands and flushing of the face were prominent in all the varieties, especially the non-paralytic and spinal paralytic groups. Extreme restlessness and insomnia occurred in 2 of the fatal cases, who also displayed marked twitching of the facial musculature. Three patients were aware that something serious was wrong with them and seemed to conserve every ounce of strength for the forthcoming battle between life and death. Two of the fatal cases were alert and conscious up to the very end, the third one lapsing into coma very shortly before death. Convulsions occurred in 2 cases. Encephalitic symptoms were thus prominent in this series and were noted in 32 (64%) of the cases, 16 of which belonged to the spinal paralytic, 9 to the non-paralytic, 4 to the bulbo-spinal and 3 to the bulbar group.

v: *Bulbo-Spinal Group* (*Six Cases*). This group included those cases showing involvement of the cranial nerve nuclei together with respiratory or circulatory centre involvement as well as paralysis of the peripheral musculature. The common symptoms were fever and stiffness of the neck and back. Paralysis was only complained of in 2 cases. Of the signs, tachycardia, pyrexia and muscle weakness were present in each case. Four cases showed muscle stiffness. The same number demonstrated diminished or absent reflexes. Hypertension was present in 3 cases. Encephalitic symptoms, viz. apprehension and tremor, were present in 3 cases. Vasomotor phenomena were only present in 2 cases. Three of the fatal cases belonged to this group. One of the remarkable features was the retention of consciousness right up to the moment of death in all the fatal cases.

vi: *Spinal Paralytic Group* (*Nineteen Cases*). The commonest symptoms were fever, spinal stiffness and weakness. Next came headache and pains in the limbs. Of the signs, tachycardia, pyrexia, muscle stiffness, apprehension and tremor along with muscle weakness in every case figured prominently. The reflexes were increased in 12 cases and decreased in 11, while localized muscle pain was complained of in 8 cases. Vasomotor phenomena, muscle tenderness and skin hyperaesthesia were present in the 10 cases. Hypertension was present in only 6 instances. The obvious difference between this group and the bulbar and bulbo-spinal form was the increased frequency of the encephalitic manifestations as well as muscle stiffness. The reason for these differences was unknown.

vii: *Non-Paralytic Form* (*Seventeen Cases*). The main symptomatology in this group comprised fever, spinal stiffness, headache and vomiting. A small number of cases, however, complained of drowsiness, convulsions and limb pain. Tachycardia, muscle stiffness, constipation, pyrexia and hyper-reflexia were the most frequent signs. Apprehension, vasomotor phenomena, muscle tenderness and skin hyperaesthesia were present in just under half the cases. Hypertension was present in 4 cases and hypo-reflexia in one case. A comparison of this group with the spinal paralytic group revealed a close similarity in most of the symptoms with the obvious exclusion of weakness. The signs, however, were also very similar, with a few notable exceptions. Hyper-reflexia was just as common in the non-paralytic group whereas hypo-reflexia was more frequently found in the spinal paralytic group. Apprehension and tremor were more prominent in the spinal paralytic group.

From these data we might conclude that the clinical features of poliomyelitis have changed very little since the turn of the century. Some important differences had, however, been noted: drowsiness had been replaced by apprehension and tremor, and the incidence of muscle stiffness in some epidemics had certainly increased.

SUMMARY

1. The detailed clinical features of acute poliomyelitis are presented. Most of the cases occurred in children under 10 years of age.

2. Prodromal symptoms occurred in 62% of the 50 cases. They comprised fever, malaise, headache and sore throat.

3. Fever, neck or back stiffness, muscle paralysis, headache and limb pain were the prominent symptoms in this series. The epidemics chosen for comparison show similar symptomatology. Drowsiness, so common in the earlier epidemics, was not a feature in this series.

4. (*a*) All the cases in this series were in excellent physical condition. Their mental rating was also higher than average. Anxiety and apprehension were noted in 30 patients.

(*b*) Tachycardia, fever, stiffness of the neck, back and hamstring musculature, paralysis, tremor and limb pain were the commonest signs.

There was no relationship between the site of the limb pain and the subsequent paralysis.

Hyper-reflexia was the rule except where paralysis involved the muscle responsible for the reflex.

Encephalitic signs, viz. anxiety, irritability, hyper-reflexia and tremor of the outstretched upper limbs, were prominent.

(*c*) Hypertension was noted in 16 cases and could be related to the anxiety of the patient, stimulation of the sympathetic component of the autonomic nervous system or be associated with hypoxia in the bulbar and bulbo-spinal groups.

(*d*) Several features of autonomic nervous system (ANS) dysfunction were present—constipation, cold clammy extremities, anxiety and hypertension.

5. Certain differences were noted in the 4 types described.

The bulbar group usually presented with paralysis. Muscle dysfunction, encephalitic symptoms and signs of ANS dysfunction were not prominent.

In the bulbo-spinal group, muscle dysfunction and encephalitic symptoms were present in half the number of cases.

All the cases in the spinal paralytic group showed paralysis, encephalitic features and muscle dysfunction.

ANS dysfunction was noted in half the paralytic cases. The non-paralytic group demonstrated encephalitic signs and muscle dysfunction in most instances and ANS dysfunction in about half the cases.

OPSOMMING

1. Die breedvoerige kliniese kenmerke van akute poliomiëlitis word beskryf. Die meeste gevalle het voorgekom by kinders onder 10 jaar.

2. Siekte-aankondigende simptome is waargeneem by 62% van die 50 gevalle. Dit het bestaan uit koors, 'n gevoel van onwelsyn, hoofpyn en seerkeel.

3. Koors, styfheid van die nek of rug, spierverlamming, hoofpyn en pyn in die ledemate was die opvallendste simptome in hierdie reeks. Die epidemies wat vir vergelykingsdoeleindes gekies is, toon 'n dergelike simptomatologie. Lomerigheid wat 'n gewone verskynsel tydens die vroeëre epidemies was, was nie 'n kenmerk van hierdie reeks nie.

4 (*a*). Al die gevalle in hierdie reeks was in 'n uitstekende fisiese toestand. Hul geestesvermoë was hoër as die gemiddelde. Besorgdheid en vrees is by 30 pasiënte waargeneem.

(*b*) Hartversnelling, koors, styfheid van die nek, die rug en die hakskeensening-spierstelsel, verlamming, bewing en pyn in die ledemate was die gewone tekens.

Daar was geen verwantskap tussen die plek waar die pyn in die ledemate ondervind is en die latere verlamming nie.

Hiperrefleksie was die reël, behalwe in die gevalle waar die spier wat verantwoordelik vir die refleks is, deur die verlamming aangetas is.

Encephalitis-tekens, nl. besorgdheid, prikkelbaarheid, hipperrefleksie en bewing van die uitgestrekte boonste ledemate, was opvallend.

(*c*) Hoë bloeddruk is waargeneem in 16 gevalle en kon in verband gebring word met die besorgdheid van die pasiënt of die stimulering van die simpatiese bestanddeel van die outonomiese senuweestelsel, of was geassosieer met hipoksie by die bulbêre en die bulbêre-ruggraatgroep.

(*d*) Etlike kenmerke van disfunksie van die outonomiese senuweestelsel (OSS) is waargeneem—hardlywigheid, koue, klam vingers en tone, besorgdheid en hoë bloeddruk.

5. Sekere verskille is waargeneem by die 4 tipes wat beskryf word.

Verlamming het gewoonlik by die bulbêre groep voorgekom. Spierdisfunksie, encephalitis-simptome en tekens van OSS-disfunksie was nie opvallend nie.

In die bulbêre-ruggraatgroep is spierdisfunksie en encephalitis-simptome by die helfte van die gevalle opgemerk.

Al die gevalle in die ruggraat-verlammingsgroep het verlamming, encephalitis-kenmerke en spierdisfunksie geopenbaar.

OSS-disfunksie is waargeneem by die helfte van die verlammingsgevalle. Die nie-paralitiese groep het in die meeste gevalle encephalitis-tekens en spierdisfunksie geopenbaar, asook OSS-disfunksie by ongeveer die helfte van die gevalle.

(*To be continued*)

THE SYNDROME OF CHEST PAIN IN HIGH GASTRIC ULCER

JULIUS BUCH, M.B., B.CH., M.R.C.P., EDIN.
Johannesburg

Shedrow[1] reported 4 cases of gastric ulcer in which there were no symptoms of gastric disturbance except for pain in the chest. It appears that pain in the chest may be the only symptom associated with a gastric ulcer lying high up on the lesser curvature. The realization that chest pain may be caused by an ulcer in this situation, with no other symptoms to suggest gastric involvement, is important from the diagnostic viewpoint. It would seem feasible that patients complaining of chest pain for which no cause can be found, may well prove to be cases of high gastric ulcer. Four illustrated cases are described in this communication.

Case No. 1. A male, aged 31 years, complained of pain across the anterior aspect of the chest and between the shoulder blades.

This was his only symptom. It had continued for 4 years. The pain had no periodicity and was unrelated to meals. It would occur spontaneously without any apparent cause. He was investigated for the chest pain and all investigations, which included, X-ray of the chest, electrocardiogram, X-ray of the cervical and dorsal spine, proved to be normal. He continued suffering in this way for 4 years. In spite of the complete absence of symptoms relating to the digestive tract, a barium meal was performed. The X-ray revealed a large gastric ulcer lying posteriorly, in close proximity to the gastro-oesophageal junction.

The patient was treated rigidly for the ulcer. The therapy consisted of antacids, anti-cholinergics and Meulengracht's diet. After 2 weeks' treatment the symptoms of pain disappeared. When the patient discontinued his treatment, the symptoms recurred. He was instructed to continue treatment, which produced immediate relief of his symptoms.

Case No. 2. A male, aged 51 years, complained of pain between the scapulae and pain in the left anterior chest in the third intercostal space. There was a history of duodenal ulcer 25 years before. The pain in the chest was the only symptom. There was no epigastric pain or tenderness. There were no symptoms of heart-burn or symptoms relating to the digestive system. The pain continued for 3 years with periods of remission lasting as long as 3 months. Recently the pain had become extreme and was controlled only by Pethidine and Morphine. The pain appeared to have no relationship to any known factor. Investigations, which included an electrocardiogram, X-ray of the chest and a barium meal, were negative. The barium meal revealed a healed duodenal ulcer.

X-ray of the dorsal spine showed a moderate degree of osteo-arthritis which was not considered to be the cause of the chest pain. The pain increased in intensity and it was decided to perform a laparotomy, with the possibility of pancreatic neoplasm in mind.

At the operation a large gastric ulcer was discovered lying on the posterior lesser curvature close to the cardio-oesophageal junction. A gastrectomy was performed. There was no evidence at the operation of pancreatic pathology. The duodenal cap showed no ulceration. The patient was immediately relieved of his symptoms and continues in good health.

Case No. 3. A male, aged 50 years, complained of pain between the scapulae. This had been present for over a year. There was no abdominal pain and there were no other symptoms relating to the digestive system. A barium meal failed to reveal any abnormality. The barium meal was repeated a month later and a large gastric ulcer was noted on the posterior wall close to the lesser curvature aspect just distal to the gastro-oesophageal junction. The patient was treated for the ulcer but has failed to report on his progress.

Case No. 4. This case history is incomplete because the exact pathology is unknown. The patient, a male aged 67 years, complained of severe pain between the scapulae and persistent annoying hiccough. He was known to have had a duodenal ulcer 5 years before. The electrocardiogram and the chest X-ray were normal. There was osteo-arthritis of the dorsal spine. The pain between the scapulae reached a severe intensity and he was admitted to a nursing home. Here it was decided to perform a further barium meal. During the barium meal X-ray the radiologist observed on the screen that the barium had perforated through the lower end of the oesophagus at the gastro-oesophageal junction, and had passed into the mediastinum. The patient died a few hours later. As an autopsy was not performed, it was not possible to state the exact cause of the lesion or its extent. The patient died of a perforation of an ulcer at the gastro-oesophageal junction. The ulcer may have been neoplastic or benign.

SUMMARY

Four cases are described in which the outstanding symptom was pain in the upper part of the chest and a minimum of symptoms relating to the digestive system.

The symptom of high chest pain should always include in the differential diagnosis the possibility of an ulcer in the region of the gastro-oesophageal junction.

OPSOMMING

Vier gevalle word beskryf waar die opvallendste simptoom pyn in die boonste gedeelte van die bors was, met minimum-simptome wat op die spysverteringstelsel betrekking gehad het.

As daar simptome van pyn hoog in die bors is, moet die differensiële diagnose rekening hou met die moontlikheid van 'n sweer in die omgewing van die maag-slukderm-aansluiting.

REFERENCE

Shedrow, A. (1957): S. Afr. Med. J., **31**, 802.

SKELETAL CHANGES IN ENDOCRINE AND METABOLIC DISORDERS

XVIII. RICKETS AND OSTEOMALACIA REFRACTORY TO ORDINARY AMOUNTS OF VITAMIN D

W. P. U. JACKSON, M.D., M.R.C.P., D.C.H.

Endocrine Clinic, Department of Medicine, Groote Schuur Hospital and University of Cape Town

Rickets (in childhood) and osteomalacia (in adults) which are relatively resistant to cure by vitamin D, are virtually either:

(*a*) Intestinal, with diminished absorption of calcium and phosphorus; or

(*b*) Renal, with excessive urinary excretion of phosphorus (and perhaps calcium).

I. *Steatorrhoea.* The usual intestinal group is formed by the steatorrhoeas, including coeliac disease in childhood (coeliac rickets), idiopathic steatorrhoea, and sprue in adults. Bone disease is much less common with pancreatogenous steatorrhoea (e.g. pancreatic fibrocystic disease or chronic relapsing pancreatitis) and chronic obstructive hepatic cirrhosis, but may be seen in the intestinal loop and stricture syndromes, and in other varieties of jejuno-ileal insufficiency. Although all these are usually classified as steatorrhoeas, it must be emphasized that the actual loss of fat in the stool is seldom really outstanding, and the general term 'malabsorption syndrome' is preferable. Gee's 'loose, unformed, not watery, very bulky, pale, yeasty, frothy, stinking, yellow or drab' stools will, however, take a lot of living down. In the syndromes mentioned, the osteomalacia is basically caused by a malabsorption of vitamin D and is corrected by parenteral calciferol.

Chemically there is a low serum phosphorus and a raised alkaline phosphatase. The serum calcium may be low (with tetany) or may be maintained by a secondary hyperplasia and hyperactivity of the parathyroid glands. Dent and co-workers[2] have actually described the appearance of parathyroid adenomas in steatorrhoea, with radiological evidence of hyperparathyroid bone disease (osteitis fibrosa) as well as of osteomalacia. In general, however, the bone disorder is purely rickets or osteomalacia, and does not differ in any way from that already described.[6] Occasionally hypoparathyroidism may be found in steatorrhoea, and rarefaction of bone may also be seen in these cases (probably osteomalacia, although on theoretical grounds osteoporosis might be expected to occur when there is protein malabsorption). In this connexion it may be noted that osteomalacia has been described with hypoparathyroidism, quite apart from steatorrhoea.

Radiological Appearances[4]. The rickets which occurs before epiphyseal fusion is precisely similar to that of ordinary vitamin D lack.

The osteomalacia is likewise similar to that seen in the vitamin D lack states, as in lactating women in China and in the 'hunger osteopathy' in Europe during World War I. The whole skeleton becomes generally decalcified, the cortex thins and fractures readily occur. The bones become soft and bend, producing such deformities as the tri-radiate pelvis with gross diminution of pelvic measurements and obstruction to labour. The vertebral bodies become more or less uniformly compressed and biconcave ('cod-fish' vertebrae).

A further feature of osteomalacia, and one which may appear before any other radiographic evidence of bone disease, is the occurrence of multiple, largely bilateral, symmetrical, radiolucent bands, zones or lines of uncalcified

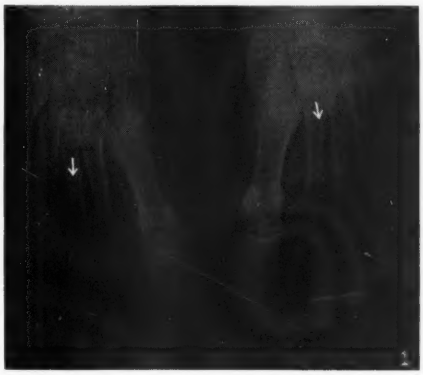


Fig. 1. X-ray of the feet of a woman aged 30 years, who had had 'idiopathic' steatorrhoea for 12 years. Serum phosphorus, 1.0 mg. per 100 c.c., serum calcium, 8.7 mg. per 100 c.c. Note demineralization and multiple translucent zones in the metatarsals.

tissue (Fig. 1). These areas are formed particularly in the ribs, the pelvis, the upper ends of the femora and humeri, and the scapulae. They vary from narrow, straight or wavy lines (which may be mere depressions in the surface or several inches long) to half-inch thick transverse bands (as in the ischio-pubic region). These 'pseudo-fractures' persist indefinitely if not treated, may increase in length, and do not stimulate callus formation unless they lead eventually to true fracture. Histologically these zones show uncalcified fibrous and cellular

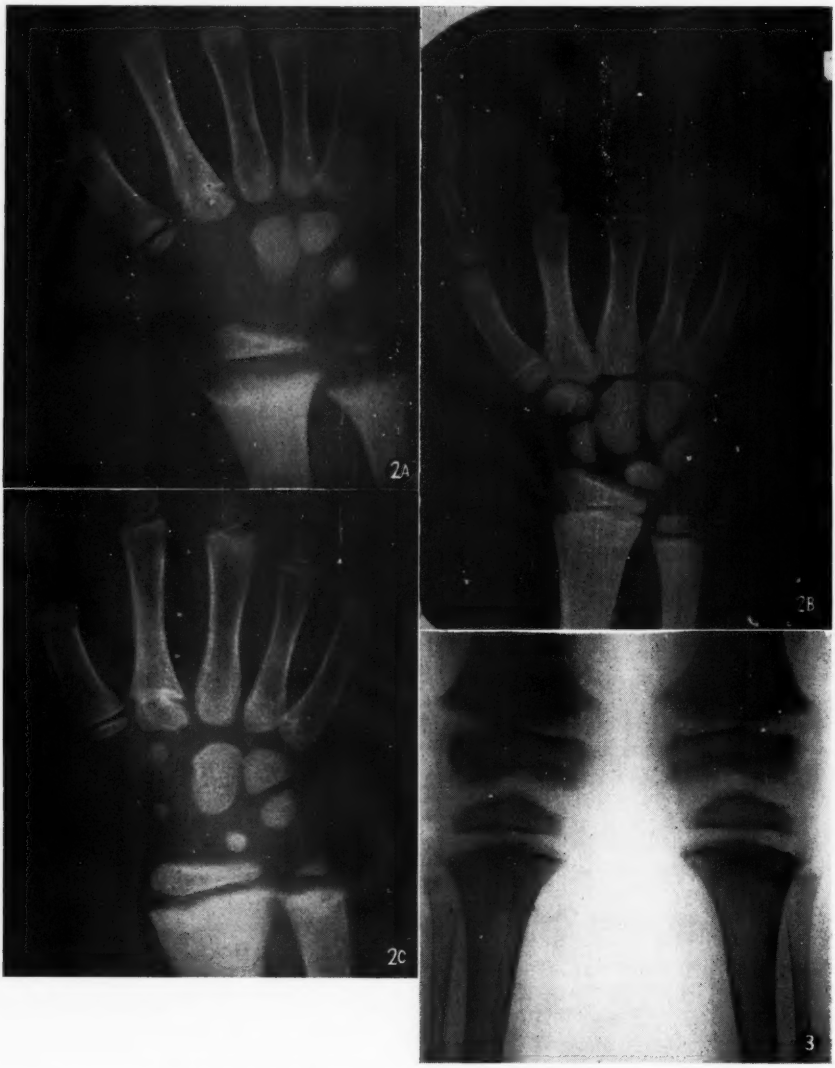


Fig. 2. X-ray of the hand of a boy of 10 years with the Fanconi syndrome. (A) Note the typical rachitic appearance of the radial and ulnar mataphyses and the delay in the osseous development compared with the appearances of a normal (control) boy of the same age (B). In (C) the remarkable improvement is shown after 10 weeks' treatment with 400,000 units of calciferol daily.

Fig. 3. Knee joints of the same case.

tissue containing many osteoblasts. Their osteomalacic nature is indicated by the large amount of osteoid tissue present. These areas are sometimes known as Looser's zones ('umbauzonen'), while the syndrome in which they appear before evident decalcification is called Milkman's syndrome.

2. *The Fanconi Syndrome*[5]. Stated briefly, Fanconi's syndrome in childhood comprises two parts:

1. Progressive deposition of cystine crystals in internal organs, including the eye and the liver, where it may be diagnostically significant; and

2. A renal tubular disorder in which a deficiency of reabsorption of phosphorus, glucose, certain amino acids and other organic acids occurs. Albuminuria, and excessive loss of fixed base (sodium, potassium and calcium) may also occur, with eventual glomerular failure and uraemia. Affected children are sickly, undersized and subject to recurrent febrile attacks accompanied by vomiting.

The disease is frequently familial, being inherited as a recessive characteristic. The ammonia-producing mechanism of the kidney is usually unaffected, and the urinary ammonia is high, as would be expected in response to the chronically acid urine. Despite the acidic urine, there is also a systemic acidosis, related to high organic acidaemia, although the serum amino acids are not elevated.

The excessive loss of phosphate into the urine (i.e. the low renal threshold for phosphorus) leads to a lowered serum inorganic phosphate. This in turn leads to rickets (Figs. 2, 3) and a raising of the alkaline phosphatase.

The same syndrome occurs in adults, except for the systemic cystinosis. The disorder is a milder one, probably on that account. In both the childhood and adult forms, actual malformation of the proximal renal tubule has been described. The rickets or osteomalacia which may be produced is precisely the same as that due to other causes. Treatment is variably unsatisfactory, but certainly large doses

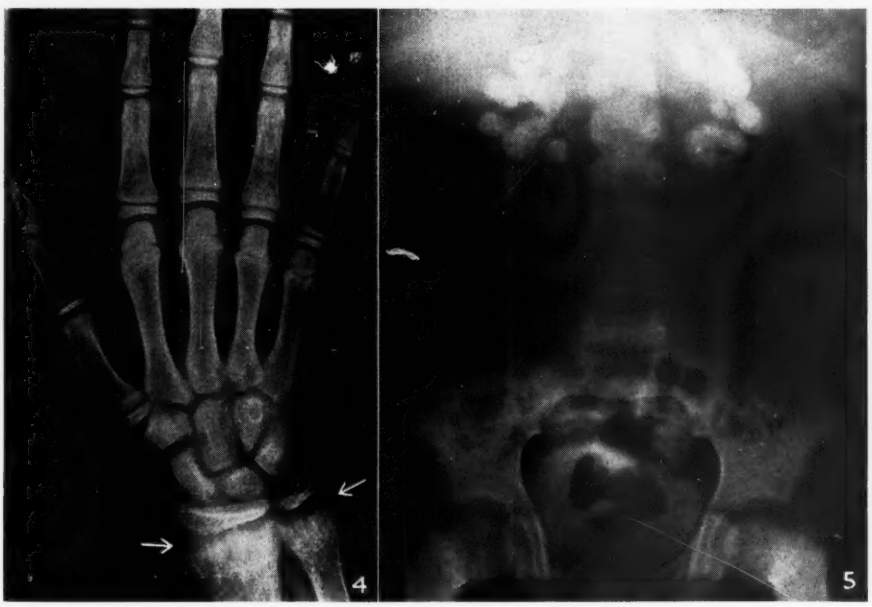


Fig. 4. X-ray of a hand in a case of renal tubular acidosis, showing typical rickets.
Fig. 5. The same case, showing dense calcification in the renal pyramids and rachitic changes in the femoral necks.
The patient was aged 13 years, with the following biochemistry:
Serum calcium: 9.0 mg. per 100 c.c.
Serum phosphorus: 1.9 mg. per 100 c.c.
Serum phosphatase: 23 units (Bodansky).
Serum chloride: 121 m Eq. per litre.
Serum CO_2: 43 volumes per 100 c.c.
The blood urea and P.S.P. tests were normal. (Case of Dr. Fuller Albright).

of calciferol will allow healing in some cases (Fig. 2C).

3. *Hypophosphataemic Glycosuric Rickets.* This condition is also, presumably, a renal tubular deficiency, (perhaps a *forme fruste* of the Fanconi syndrome) with renal glycosuria and a heavy urinary phosphate loss, but without the other features.

Milkman's original case of his syndrome probably belonged to this group.

4. *Renal Tubular Acidosis* (*Hyperchloraemic Nephrocalcinosis*). This is quite a different renal tubular abnormality with the following features:

1. Inability to acidify the urine beyond about pH 6.5.
2. A high urinary bicarbonate and a low ammonia content.
3. Systemic acidosis, with a low plasma bicarbonate and high chloride levels.
4. An excessive loss of calcium and potassium in the urine.
5. A lowered serum phosphorus, a raised alkaline phosphatase and osteomalacia.
6. Calcification of the renal parenchyma and calculus formation.

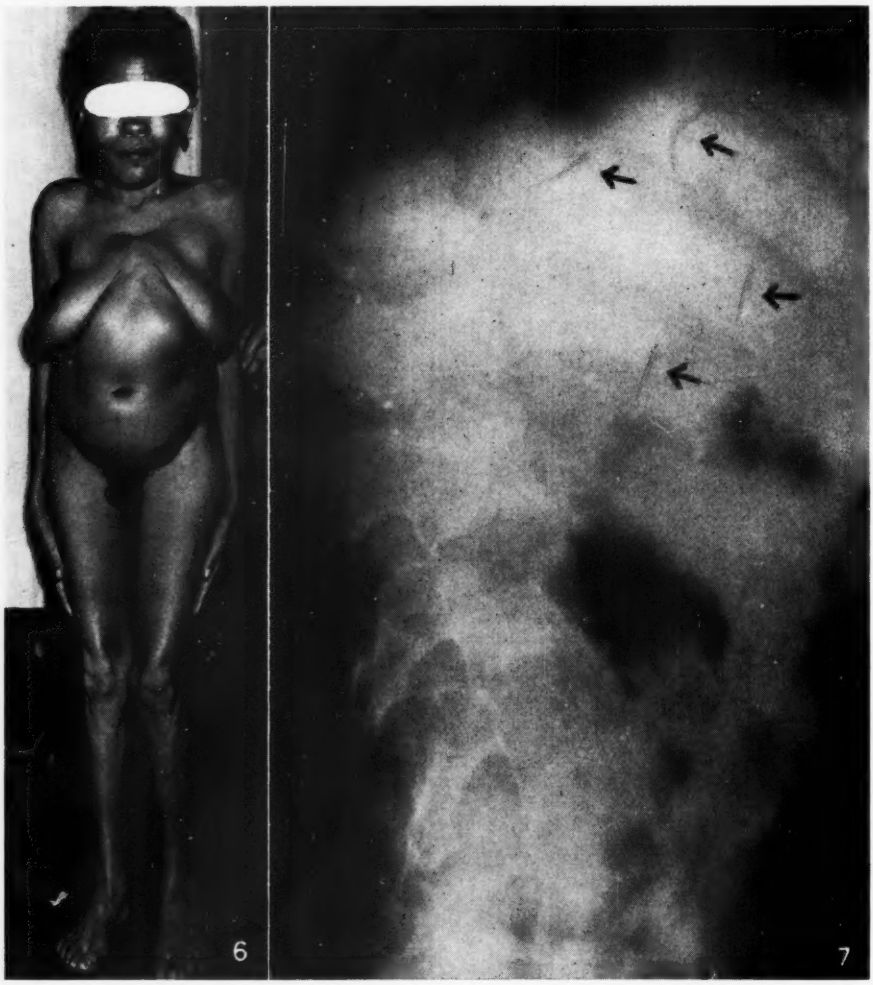


Fig. 6. A female of 32 years with vitamin D resistant osteomalacia and dwarfing. Note the poor muscular development, the deformed chest and genu valgum.
Fig. 7. An X-ray of the lateral thoracic region of the same patient. The vertebral bodies are so faint as to be almost invisible and there are several Looser's zones in the ribs (arrows).

7 Eventually pyelonephritis and glomerular failure also occur.

Albright *et al.*[1] suggest that the loss of urinary calcium tends to reduce the serum calcium. This stimulates the parathyroids and so increases the urine phosphorus and maintains the serum calcium (as in the malabsorption syndrome). A fall in serum phosphate results. The real cause of the osteomalacia in this syndrome has not been fully elucidated. In fact, the basic mechanism of the whole disease process remains obscure, although it has been claimed to be an inability of the tubules to reabsorb bicarbonate ion, such as might occur if the tubular enzyme, carbonic anhydrase, were absent. Diamox, which inhibits carbonic anhydrase, does produce some of the biochemical features of the disease, but the effect is not maintained.

Radiological features (Figs. 4, 5) include those usual in osteomalacia, together with varying degrees of nephrocalcinosis and renal calculi. The most successful treatment includes the alkalinizing agent, sodium citrate, which presumably acts by correcting the systemic acidosis and producing base for the urinary excretion of acid.

5. *Vitamin D Resistant Rickets and Osteomalacia*[3]. In some children rickets occurs and progresses despite a normal diet and the addition of the usual amounts of vitamin D, and in the absence of steatorrhoea or renal disorder. This type of rickets is frequently inherited as a Mendelian dominant characteristic. It can only be diagnosed in single cases by exclusion of other causes for rickets.

As usual, the serum phosphorus is low and the alkaline phosphatase is raised. Unlike ordinary rickets, however, the serum calcium is apparently never markedly depressed, so that tetany does not occur. The urine calcium may be normal or very low indeed.

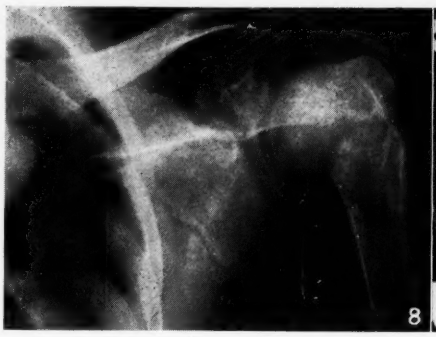
Because of the virtual impossibility of adequate therapy, the sufferers remain dwarfed and deformed in adult life (Fig. 6) and may even retain their chemical and histological osteomalacia, although they lose their bone pains and are quite strong.

Much more rarely, a similar condition starts in adolescence or early adult life. It is uncertain whether this is really a separate disease entity, although it is my own opinion that there are several different conditions at present masquerading under the single head of 'resistant rickets and osteomalacia'. Thus it is uncertain whether the basic aetiology is one of excessive phosphate loss caused by inability of renal tubular reabsorption (as in the Fanconi syndrome) or one of refractoriness to vitamin D in its calcium-absorbing activity (i.e. an intestinal variety of osteomalacia).

In actual fact there seem good arguments for and against either view (these become very involved) and it seems highly probable that both aetiologies, together or severally, may play a part in different cases.

Radiologically, once again the typical features of rickets and osteomalacia are found (Figs. 7–9). In the adult case, the bony trabecular pattern becomes remarkably coarse, although this is no different from the appearance following any very longstanding rickets.

The only specific treatment consists of calciferol in enormous doses, which may need to be in the region of a million units daily. Overdosage, with vitamin D poisoning, is quite close to the curative dose, so that a rise in serum or urinary calcium to abnormally high



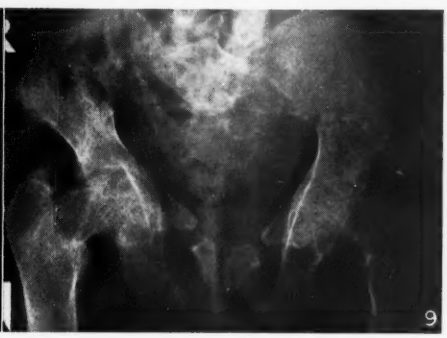


Fig. 8. The shoulder girdle of the same case. Note Looser's zones in the scapula and the surgical neck of the humerus.
Fig. 9. The pelvis of the same case showing the broad decalcified bands in the ischio-pubic bones, narrower zones radiating from the pelvic brim, the mis-shapen pelvis and coxa vara.

levels must be watched for. Dihydrotachysterol (AT10) may also be curative.

6. Other Causes. Other rare causes of rickets and osteomalacia include chronic nephritis or glomerular disease in childhood (which produces features of rickets in addition to osteitis fibrosa), possibly idiopathic hypercalciuria, hyperthyroidism, osteopetrosis, the recovery phase after parathyroidectomy for hyperparathyroidism with bone disease, and hypophosphatasia. These will not be further considered here.

OPSOMMING

Die soorte weerspannige rhachitis en osteomalakie wat veroorsaak word òf deur ingewandsabsorberende òf nierbuisdefekte word oorweeg. Die toestande sluit veral in steatorrhee, Fanconi se sindroom, nierbuisasidose en sogenaamde idiopatiese vitamien-weerstandskragtige rhachitis.

Die radiologiese kenmerke van osteomalakie word beskryf as iets wat al hierdie vorms gemeen het en sluit die sogenaamde ‚melkman se sindroom' in.

Die verskillende meganismes wat beenletsels in al die afsonderlike gevalle veroorsaak, word oorweeg.

REFERENCES

1. Albright, F., Burnett, C. H., Parson, W., Reifenstein, E. C. and Roos, A. (1946): Medicine, **25**, 399.
2. Davies, D. R., Dent, C. E. and Willcox, A. (1956): Brit. Med. J., **2**, 1133.
3. Dent, C. E. and Harris, H. (1956): J. Bone Jt. Surg., **38B**, 204.
4. Fairbank, T. (1951): *Atlas of General Affections of the Skeleton.* London: E. & S. Livingstone.
5. Jackson, W. P. U. and Linder, G. C. (1955): Quart. J. Med. N.S., **22**, 133.
6. Jackson, W. P. U. (1957): Med. Proc., **4**, 149.

CRANIOSTENOSIS, ASYMMETRY AND GROWTH OF THE SKULL

HARRIS JACKSON, L.R.C.P., M.R.C.S., D.M.R.
Johannesburg

(*Continued from p.* 228)

CRANIAL PATHOLOGY: DEFICIENT GROWTH

So far only intracranial causes of asymmetry of the skull have been considered. The next case illustrates a cranial cause due to premature synostosis of the left half of the coronal suture, with consequent absence of growth.

A CASE OF PLAGIOCEPHALIC CRANIOSTENOSIS

A 23-year-old woman was X-rayed in a routine examination. There were no symptoms or signs of intracranial pathology. The left hemicranium was smaller, with deeper and more numerous convolutional impressions and a thinner vault (1, 3 and 2 in Fig. 23). The lesser wing was markedly elevated and oblique, and the petrous temporal slightly depressed (9 and 11 in Fig. 23). The left orbit was larger, and the linea innominata normal. The left half of the coronal suture was absent, but the other sutures were visible. The sagittal suture was vertical. The groove for the superior sagittal sinus was displaced to the right and the crista galli was in the mid-line. The basal view showed a very small left anterior fossa, due to considerable backward displacement of the frontal squame and to slight forward displacement of the anterior wall of the middle fossa (A′ and B′ in Fig. 24). In the lateral view (Fig. 25) again only one side of the coronal suture was visible. The exaggeration of the convolutional impressions anteriorly was demonstrated.

Plagiocephaly from localized craniostenosis is said to be rare, and to be due to involvement of one temporo-parietal suture.[19] The only cases I have personally encountered or seen reported involved one half of the coronal suture.[11]

The diagnosis may be difficult unless one understands how the pattern is produced. Absence of growth at the suture leads to a small anterior fossa which becomes closely adapted to the underlying growing brain. This gives the picture of a small hemicranium, particularly the anterior fossa, with increased convolutional impressions on, and thinning of the frontal squame. The coronal suture on the same side is then found to be absent (Fig. 26).

CRANIAL PATHOLOGY: EXCESSIVE GROWTH

A CASE OF ASYMMETRY WITH HEMIVERTEBRAE

This child was referred for congenital torticollis due to numerous irregularly fused cervical and cervico-dorsal hemivertebrae. The skull was grossly asymmetrical. There were no sym-

toms or signs of intracranial pathology. The sphenoidal fissure view (Fig. 27) shows a bizarre asymmetry. The left side is enlarged and slightly thickened and its convolutional impressions are less well developed. The sagittal suture is oblique (*4a* in Fig. 27). The left coronal suture contains a large accessory bone inferiorly (arrows in Fig. 27). Pneumatization is normal, with the frontal sinuses larger on the right and the ethmoids larger on the left. The left orbit, lesser wing of sphenoid and petrous temporal are elevated. A satisfactory basal view could not be obtained because of the unusual asymmetry of the head.

Although asymmetry of the skull occurs with torticollis and with scoliosis, this case did not conform to the pattern of scoliosis capitis (*vide infra*). The unusual asymmetry was quite unlike that due to extracranial pressure and, because of the presence of hemivertebrae (some of which were supernumerary), it is assumed that the accessory bone in the coronal suture was the site of active growth, although the usual sutural bone (which is symmetrical) does not produce asymmetrical growth.

DISPLACEMENT OF THE LESSER WING OF THE SPHENOID

In this paper great emphasis has been placed on the position of the lesser wing of the sphenoid. It is one of the most useful and interesting anatomical features of the skull and it has great diagnostic significance. It may be elevated or depressed, though the higher wing more commonly indicates the pathological side. Unilateral elevation was once regarded as pathognomonic of a chronic subdural haematoma, but this has been disproved. Bull predicted it could occur with a temporal lobe tumour, and many other causes have since been found (Table I). The position of the lesser wing appears to be affected mainly by growth

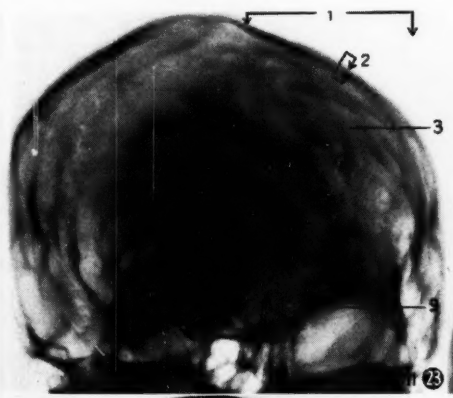


Fig. 23

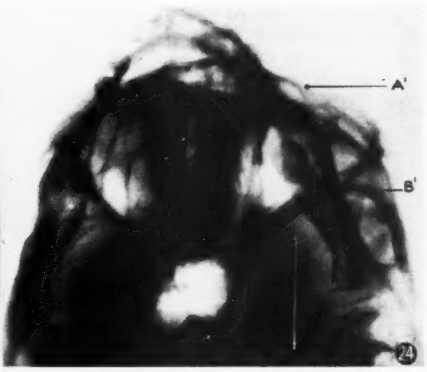


Fig. 24

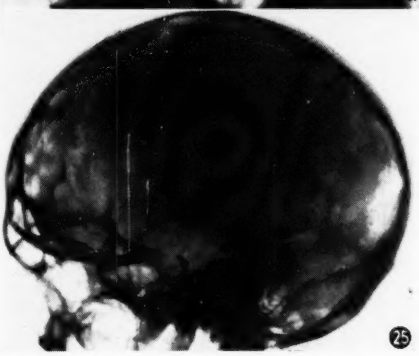


Fig. 25

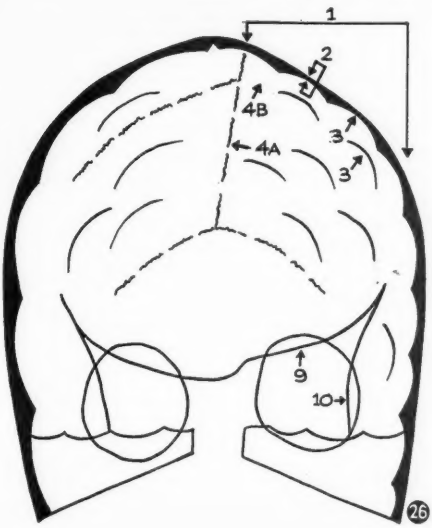


Fig. 26

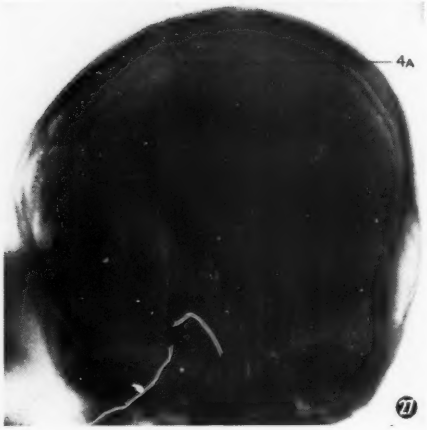


TABLE 1: DISPLACEMENT OF THE LESSER WING OF THE SPHENOID

i. *Intracranial Mass.*
Chronic subdural haematoma or hygroma.*
Leptomeningeal cyst.*
Tumour* or gliosis.
Aneurysm.*
Neurofibromatosis.*
ii. *Cerebral Atrophy* and Sturge-Weber Syndrome.
iii. *Cranial Pathology.*
Craniostenosis.
Active sutural bone.
Maldevelopment of wings of sphenoid.
iv. *Eye.*
Agenesis.
Calcified lens.*

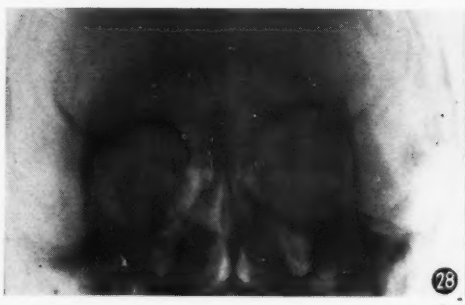
*Previously reported.[2, 3, 5]

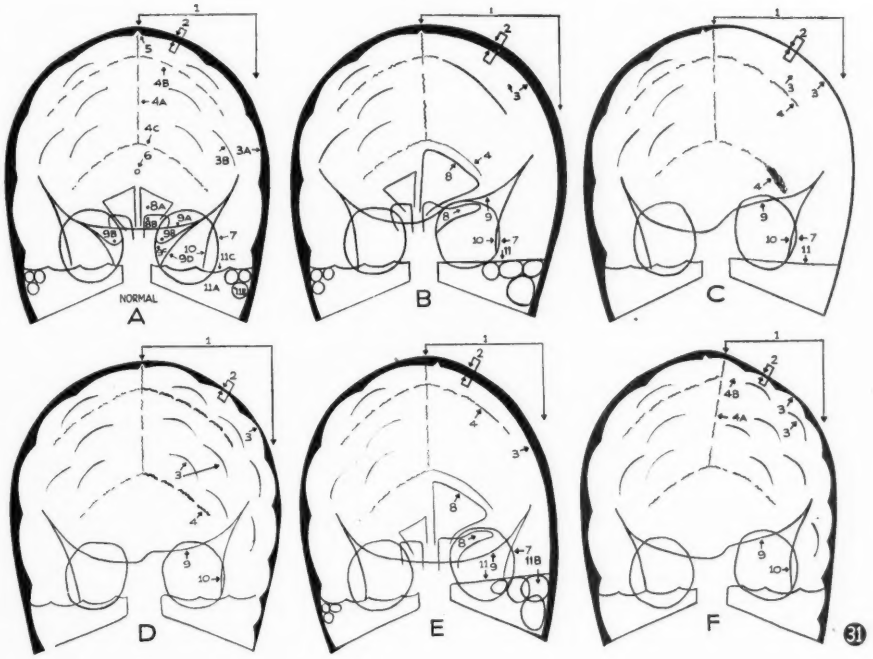
(Note that bone pathology such as fibrous dysplasia, Paget's disease etc. may result in asymmetry).

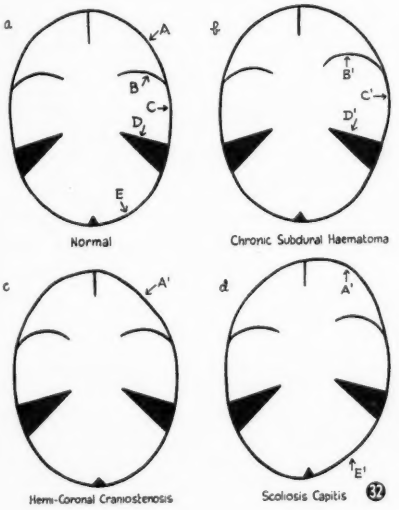
at the temporo-parietal suture. To complete the picture, two further causes of lesser wing displacement are demonstrated. Fig. 28 is from a case of neurofibromatosis in which, in addition to elevation of the lesser wing, there is maldevelopment of the whole orbit. The cause of this is not known. Fig. 29 shows depression of the lesser wing due to underdevelopment of the orbit secondary to agenesis of the eye.

ASYMMETRY OF EXTRACRANIAL ORIGIN

A common type of asymmetry of the skull has been described as scoliosis capitis by the orthopædic surgeons, and it is also well known to pædriatricians. In the sphenoidal fissure view this type of skull shows no definable asymmetry, but the basal view is characteristic (Fig. 30). The skull appears to be rotated about its sagittal plane, so that the whole of one side lies







Figs. 31 *and* 32. The appearances of the cranial asymmetries have been summarized in Tables 2 and 3 (p. 360), and are shown diagramatically in Figs. 31 and 32.

anterior to the other, producing a rounded rhomboid shape. The term rhomboid skull has been suggested by Fainsinger.[21] The forehead bulges on one side and the corresponding side of the occipital squame is flattened. The hair over this flattened part may be sparse or absent in the young infant. This asymmetry appears to be due to the infant's favouring one side when lying on its back so that the skull moulds asymmetrically as the result of external stresses. These may be exogenous (due to pressure, gravity) or endogenous (due to muscle stresses). A soft, malleable skull, e.g. of rickets, fragilitas ossium, favours its development, but it must be remembered that malleable in this sense does not mean simply easily distorted by stresses, but growing easily into a shape corresponding to distortion that would be produced by such stresses. The incidence of this asymmetry is high in the presence of pathology, e.g. in mentally retarded infants who fail to sit at the proper age; but it is common enough in perfectly normal people. It neither indicates nor precludes the presence of intracranial pathology.

TABLE 2: SPHENOIDAL FISSURE VIEW, FIG. 31

	In the Vault				*In the Base*			
	Size	*Thickness*	*Convolutional Impressions*	*Sutures*	*Pneumatization*	*Lesser Wing*	*Greater Wing*	*Orbit*
Reference No. in Fig. 31	1	2	3	4	8	9	10	7
Chronic subdural haematoma (early) (C)	Increased	Decreased	Decreased or absent	Widened	?Inhibited	Elevated	Displaced laterally	Elevated
Chronic subdural haematoma (late) (B)	Increased	Increased	Decreased or absent	Normal	Increased	Elevated	Displaced laterally	Elevated
Tumour or infiltration (D)	Increased	Decreased	Increased	Widened	Normal	Elevated	Normal or displaced laterally	Normal
Cerebral atrophy (E)	Decreased	Increased	Decreased or absent	Normal or early fusion	Increased	Varies	Normal	Elevated or normal
Sturge-Weber syndrome	Decreased	Varies	Varies		?	?	Normal	?
Hemi-coronal craniostenosis (F)	Decreased	Decreased	Increased	Absent half of coronal	Normal	Elevated	Normal	Normal
Scoliosis capitis	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal

TABLE 3—BASAL VIEW (FIG. 32)

	Anterior Wall of Anterior Fossa	*Anterior Wall of Middle Fossa*	*Posterior Wall of Posterior Fossa*
Reference No. in Fig. 32	A	B	E
Chronic subdural haematoma (b) ..	Normal	Forwards	Forwards
Hemi-coronal craniostenosis (c)	Backwards	Normal	Normal
Scoliosis capitis (d)	Forwards	Forwards	Forwards

(*To be continued*)

NOTES AND NEWS : BERIGTE

Dr. J. Graham Scott of Johannesburg, a member of the Committee of the Bureau for the Prevention of Blindness (South African National Council for the Blind), will represent the National Council in Brussels, at the International Congress on Ophthalmology.

* * *

Dr. O. M. Haarburger of Cape Town is on a 5-month trip to Europe and England. During his stay overseas he will visit clinics in Austria, Switzerland and Holland, and will attend the International Ophthalmological Congress in Brussels.

Medical Distributors (Eiens.) Bpk., van Johannesburg, het hulle intrek geneem in 'n moderne en ruim perseel te Jeppestraat 252, waar hulle die helfte van die eersteverdieping van „CAPE-YORK," 'n imposante nuwe gebou wat strek van Goud tot Nuggetstrate, bewoon.

Die ingang is op die grondverdieping (derde winkel vanaf die hoek van Goudstraat). Aantreklike trappe, die mure langsaan versier met kunswerke deur Ernest Ullmann en tonele van die Mediesegeskiedenis, neem die besoeker tot in die moderne en ruim kantore.

Al die ou en nuwe vriende van Medical Distributors word vriendelik uitgenooi om ons nuwe perseel te kom besigtig. Parkeerplek sal beskikbaar wees in die kelder sodra die gebou voltooi is. In die tussentyd vra Medical Distributors hulle besoekers om die ‚Parkeerknoppie' in die ingangsportaal te druk sodat hulle karre veilig parkeer kan word.

Die Johannesburg Hoofkantoor is onder die persoonlike toesig van mnr. H. J. Kretschmer wie reeds vir meer as 20 jaar verbonde is aan die mediese en chirurgiese handel.

Die Kaapstadse Agentskap van *Dismed* is in die hande van mnr. H. E. Lewy, Bostonhuis 216–217, Strandstraat, wie welbekend is aan praktisyns in die Kaapprovinsie.

* * *

DR. GAVIN HILDICK-SMITH

Dr. Gavin Hildick-Smith, who spent his early youth in South Africa and later returned to serve in two Johannesburg hospitals, is the new Associate Director of Clinical Research for Johnson and Johnson, at the company's Research Centre in New Jersey, U.S.A.

From 1948 to 1950 he served at Baragwanath Hospital and at the Infectious Fevers Hospital (non-European) in Johannesburg.

In addition to his work at the hospitals in Johannesburg, he has served at hospitals in London, Philadelphia, Pennsylvania, and at Ottawa and Toronto in Canada. He also was a lecturer at the University of Ottawa.

Born in South Africa, Dr. Hildick-Smith left Johannesburg at the age of 10 to be educated in England. He later studied at Cambridge University and became a Member of the Royal College of Physicians in 1952. In 1954 he received his Doctorate of Medicine from Cambridge University and that same year became a Diplomate of the Pediatric Boards of Canada.

At Johnson and Johnson, Dr. Hildick-Smith will assist Dr. John Henderson, Medical Director, in the field of clinical research.

* * *

HONORARY DEGREES AWARDED BY THE UNIVERSITY OF NATAL

TO PROF. S. F. OOSTHUIZEN AND DR. A. B. TAYLOR

At the Graduation Ceremony of the University of Natal, held on 29 March 1958, Prof. S. F. Oosthuizen, President of the South African Medical and Dental Council, was awarded the degree D.Sc. *Honoris Causa*, and Dr. A. B. Taylor was awarded the degree of Ph.D. *Honoris Causa*.

The following citations were presented by the University Orator, Prof. S. F. Bush on the occasion of the awards.

SAREL FRANCOIS OOSTHUIZEN

D.Sc. (*Honoris Causa*)

To-day the University of Natal proudly welcomes to full membership the first graduates in the Faculty of Medicine. Each of them has already declared that he will 'exercise his profession for the good of all persons whose health may be placed in his care, and for the public weal.' And so they become the latest to join the long line, stretching back through the ages, of those who, whether primitive witch-doctor or modern medical practitioner, honourably strive to relieve human suffering and to ensure the greater health and welfare of their fellow-men.

But if you would know which doctor is witch, and which the wizard of modern medical science and practice, ask Dr. Oosthuizen, for it is the South African Medical and Dental Council which holds the Roll of those who, having satisfied the exacting academic and professional standards of modern medical education and discipline, are qualified to assume the 'obligations of the medical profession' in South Africa, and follow in the 'honourable traditions' prescribed, two thousand years ago, by Hippocrates, the father of modern medicine.

Sarel Francois Oosthuizen has been President of the South African Medical and Dental Council for 7 years, and has exercised a notable influence upon the development and established reputation of the Medical Faculty, from the moment when the first students entered the School, to this day when they become the University's first medical graduates.

In his own academic and professional fields of medicine, and as Professor of Radiology in the University of Pretoria, he has insisted upon and attained the highest qualifications and awards. As President of the Medical Council, a body charged with the control of medical education, and with the maintenance of standards of professional practice and ethics in this country, he has insisted that only the very highest and best are worthy of recognition, in the Medical Schools, in the hospitals and in the work of the general practitioner, irrespective of race or colour, or the financial circumstances of doctor or patient. His outstanding scientific attainments, and his unusual ability as organizer and administrator, have enabled him to lay the impress of his own worth upon all branches of medicine in this country.

Little wonder then that his colleagues have selected him as their chosen leader, or that more than one Cabinet Minister has turned to him for guidance and advice; as have also the Council for Scientific and Industrial Research, the Atomic Energy Board, the Council of his own University, and other bodies. Three times he has led South African Delegations to International Congresses on Radiology, and he has been selected as the official delegate of the Union of South Africa to numerous other International Congresses, such as the First Empire Scientific Congress in London in 1946, and the International Congress on Medical Education held in London in 1953.

And still there remain time and energy to make contributions to knowledge in various branches of medicine by researches conducted through his own special field of radiology.

Mr. Chancellor, the University has no doubt about which doctor stands now before you. Here is a very wizard of modern medical science and practice, a worthy leader of his colleagues, and a valued friend of our own Faculty of Medicine.

ALAN BOARDMAN TAYLOR

Ph.D. (*Honoris Causa*)

In 1921, a young American doctor and his wife, a Canadian nurse, arrived in South Africa, on the invitation of the late Dr. James B. McCord to share in the work of the Zulu Hospital, founded 10 years before on its present site on the Berea, and to assist in establishing in Durban a Medical School for the training of young Zulus in medicine.

That young medical man was Alan B. Taylor. He came out for 7 years, and if by then the Medical School showed no signs of materializing, he would reserve the right to reconsider his position and withdraw. It was not until 1939 that the first class of 4 African medical aides came to McCord's Hospital for their final year of training in clinical medicine, after completing the previous 4 years of training at the South African Native College at Fort Hare. Only

to-day has the first class of fully qualified non-European doctors, trained in the Durban Medical School, presented itself for graduation in the University. But Dr. Taylor has remained, for 37 years now, to serve the Zulu people, and the medical needs and aspirations of the Bantu peoples of Southern Africa. For him, as for so many others who have participated in many different ways in the founding of the Medical School, but particularly for Dr. Taylor and his wife who has shared his labours, to-day must surely be a day of fulfilment—fulfilment of the hope and promise that was in their hearts when they first set foot on the shores of Natal.

It is worthy of the man whose daily life has been devoted to the bodily and spiritual healing of the Zulu people, that he should find ' real satisfaction in opening doors for non-Europeans that have no names attached to them.' Three years after his arrival in the country, he sought and obtained Government recognition of the McCord Hospital as the first school for the training of African nurses who, on graduating, could become registered nurses. Other doors were opened in quick succession. The first African midwives to be registered in South Africa were trained at McCord's; whose graduates, too, were, as the result of Dr. Taylor's ' amiable and tireless persistence,' the first non-Europeans in Natal to gain the right to be employed as District Nurses and Midwives, and to receive senior appointments as Staff Nurses and Sisters.

It was at McCord's, under Dr. Taylor's direction, that the medical aides received their final year of training, that non-European medical graduates from South African universities first served as interns, and received appointments as Senior Medical Staff and Honorary Staff.

At last, in 1951, the doors of the Durban Medical School were opened. Dr. Alan Taylor was Chairman of the Action Committee set up jointly in 1944 by the Natal Coastal Branch of the South African Medical Association and the Natal University College to achieve the fulfilment of this purpose; and of the Committee which prepared building—and staffing—plans for the new School. In August 1950, the Council of the University appointed him the first Dean of the Faculty of Medicine. He is still a member of the Faculty Board, and holds appointment on the teaching staff as Honorary Lecturer in Obstetrics.

Dr. Taylor's many and varied activities in medical and missionary services in South Africa, and in the educational and social life and aspirations of the African and Indian peoples of South Africa, cannot be detailed here; but they will not be forgotten.

His name is perpetuated in the Alan Taylor Residence of the University and, with that of his senior partner of earlier days, in the Alan Taylor wing of the McCord Zulu Hospital. Dr. Taylor and his wife, Mary, live in the hearts and lives of the Zulu people and in the warm affection and deep respect of all his fellow-men. It is the wish of the University that he should now be the first to receive the highest award in Medicine, for which provision is made in the Statute.

* * *

RISTOCETIN†

At the Fifth Annual Symposium on Antibiotics, U.S.A., there were several reports on the recently announced antibiotic ristocetin.*

Drs. Monroe J. Romansky and J. Robert Holmes of George Washington University, told the *Symposium* of 6 patients with enterococcal and one with staphylococcal endocarditis successfully treated with ristocetin. Four of the patients had received vigorous multiple antibiotic therapy for 1, 2, 3 and 14 months before the administration of ristocetin, 2 patients had received other antibiotics for 4 and 9 days, and the seventh patient none at all.

In 5 of the 6 patients with enterococcal endocarditis the microorganism was found to be resistant to penicillin and streptomycin, and sensitive to 0.1 to 0.3 units per ml. of ristocetin. The organism from the sixth patient was sensitive to penicillin and resistant to streptomycin. According to the investigators this organism was not available for *in vitro* tests with ristocetin.

In the patient with staphylococcal endocarditis the microorganism was sensitive to several antibiotics in addition to ristocetin.

In the 7 patients the period of ristocetin therapy ranged from 10 to 24 days (average duration 16 days) and about 2 g. per day, given intravenously in 2 doses, for about 2 weeks, appeared to be adequate therapy.

A satisfactory clinical and bacteriological response was observed in the patients, the longest follow-up being 18 months.

The usefulness of ristocetin in the treatment of antibiotic-resistant staphylococcal pneumonia was pointed out by Dr. G. L. Calvy of the U.S. Naval Hospital in New York.

He told of 4 critically ill, near-moribund patients with staphylococcal pneumonia who were given large doses of other antibiotics without satisfactory response. Sensitivity tests revealed resistance to all that were used.

After ristocetin was put to use, clinical improvement was noted in each case with a temperature fall by lysis, beginning resolution of pneumonia; general improvement in sense of well-being followed directly, within 24 to 48 hours. One patient with a lung abscess responded promptly with general improvement and a fall of temperature from 104°F. to 99°F. in 12 hours.

Ristocetin was given intravenously with gradual tapering of dose during courses of therapy that were continued as long as one month. Since continuous intravenous feeding was required, the medication was injected into the tubing. No untoward reactions were noted.

Dr. Boris A. Shidlovsky, Harlem Hospital, reporting on a study to determine the effect of ristocetin on the aerobic intestinal flora of Man, stated that the antibiotic was given to hospitalized patients in 2 oral doses. The lower gave inconsistent results in inhibition of microorganisms, while the higher dosage depressed numerically the aerobic gram-positive flora in the stool. Some gastro-intestinal side effects of a minor character were encountered in the investigation.

Two forms of ristocetin, A and B, have been isolated from the fermentation broth of *Nocardia lurida*. They are essentially similar in their properties, except that ristocetin B is 3 to 4 times more potent than ristocetin A.

Further studies have revealed no development of resistance to either antibiotic. The two were additive in effect in mouse protection tests, and after prolonged daily intravenous administration to rabbits and dogs at relatively high doses, no abnormal laboratory or histological findings attributable to either of them were obtained.

† Annotation (1958): This Journal, **4**, 21.
* Spontin (Abbott).

IN MEMORIAM

JACK GREENSTEIN, M.D. (HONS.), B.CH., L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLAS.)

Dr. Jack Greenstein died in Johannesburg on Easter Monday, 7 April 1958. He was only 57 years old.

Jack Greenstein commenced his medical education at the University of the Witwatersrand, and thereafter continued at Edinburgh University, where he qualified in 1925.

After qualifying he established himself in Lichtenburg (Western Transvaal), where he built up an extensive practice, with a reputation far beyond the borders of the district. He contributed a great deal to the cultural life of that area.

In 1938 he went to England, and studied at the British Postgraduate School of Medicine, Hammersmith, where he undertook extensive investigations into the incidence and causation of thrombosis and pulmonary embolism.

He returned to Johannesburg after the outbreak of War, and accepted an appointment as District Surgeon and Lecturer in Forensic Medicine at the University of the Witwatersrand. This enabled him, with the permission and encouragement of the then Head of the Department, Professor McIntosh, to continue his investigations into circulatory disease. He did a great deal of valuable work and published several papers on his researches, among them *Thrombus Formation in the First Part of the Aorta* and *Coronary Embolism: Its Medico-Legal Aspect* and *Comparative Causes of Sudden Death in the European and the Bantu.*

In 1945 he returned to Britain for further study, and was awarded his M.D. with Honours at Edinburgh University for a thesis based on his earlier studies, viz. *Deep Vein Thrombosis and its Relation to Pulmonary Embolism.*

He next spent a year in the United States pursuing his studies in cardio-vascular disease at the Mayo Clinic, the Mount Sinai Hospital and other well-known institutions, where he developed a special interest in phonocardiography.

On his return to South Africa he joined the staff of the newly established Cardiac and Peripheral Vascular Diseases Clinics at the Johannesburg Hospital, where he applied his knowledge of phonocardiography, and recorded a survey of 1,500 cases.

In 1946 he commenced practice as a Consulting Physician in Johannesburg.

Dr. Greenstein was Senior Physician to the Discoverers Hospital on the West Rand, and on the staff of the Cardiac and Peripheral Vascular Diseases Clinics of the Johannesburg General Hospital.

He was an indefatigable worker, a shrewd observer and a clear thinker. He had very wide interests, particularly in literature and music.

Jack Greenstein had a warm, genial and lovable personality. He was a desirable friend and a wise, painstaking and thorough physician. His opinions were based on deep wisdom and understanding in addition to wide reading, and the critical evaluation of new sources of knowledge. He practised in the spirit of *Man is born to live, not for himself, but for others.*

I, personally, in the role of patient, have much cause to be eternally grateful to him for the devoted manner in which he applied himself unsparingly on behalf of his patients. I shall never forget his distress when the prognosis looked rather gloomy; how he explored every avenue of treatment, inviting every colleague who might be of assistance and the magnificent manner in which everyone responded to his call.

He will be missed by many who will retain the memory of his sincerity and modesty. An incident at the Centenary of the American Medical Association, which he attended, will illustrate this. Jack Greenstein was listening to a lecture in the Cardio-Vascular Section. He had arrived after the lecture had begun. The lecturer was quoting statistics, and Greenstein was struck by the remarkable fact that the lecturer's figures were identical with his own. It was only some time later that he discovered that the figures quoted were, in fact, Greenstein's.

Our heartfelt sympathy goes out to his widow, Bessie, and to his children, Dr. Adrian Greenstein, his daughter, Mrs. Marna Shapiro (wife of Dr. Sydney Shapiro), and to Felicity and Robert.

J. S. ZIDEL.

Johannesburg.

PREPARATE EN TOESTELLE

FUNGIZONE VIR INFUSIE

Fungizone is 'n kragtige antibioticum. Die geneesheer wat van plan is om hierdie middel te gebruik, moet volkome op hoogte van die eienskappe daarvan wees.

Fungizone is Squibb se amfoterisien B, 'n nuwe swambestrydende antibioticum. Vir binne-aarse gebruik word *Fungizone* beskikbaar gestel in die vorm van 'n steriele, geliofiliseerde poeier in flessies wat 50 mg. amfoterisien B en 'n totaal van ongeveer 46 mg. natriumdesoksicholaat met natriumfosfate as buffers verskaf. Vir toediening deur binne-aarse infusie moet die droë *Fungizone*-poeier in Dekstrose-inspuiting 5% U.S.P. opgelos word.

Amfoterisien B word verkry van 'n ongeïdentifiseerde *Streptomyces*-soort wat in 'n bepaalde Suid-Amerikaanse grondsoort aangetref word. Die afsondering, kristallisasie en biochemiese karakterisasie van die antibioticum is bewerkstellig in die laboratoriums van die Squibb-instituut vir Mediese Navorsing. Aangesien kristallyne amfoterisien B feitlik onoplosbaar in waterige stowwe is, is 'n amfoterisien-B-natrium-desoksicholaat-mengsel—'n oplosbare samestelling—ontwikkel. Kliniese toetse het bewys dat *Fungizone* 'n doeltreffender effek op 'n groter verskeidenheid van diepgesetelde swam- en sûurdeegsoorte het as enige ander swambestrydende middel wat tans vir die behandeling van menslike kwale gebruik word. Die antibioticum het geen effek op bakterieë nie. Daar is bewys dat die bloedpeile wat deur die antibioticum teweeggebring is, bly voortbestaan gedurende 'n tydperk van 18 uur nadat die binne-aarse infusie daarvan gestaak is. Dit het die mening laat ontstaan dat die antibioticum stadig deur die niere afgeskei word.

Bewaring: Flessies bevattende droë *Fungizone*-poeier moet in 'n koelkas bewaar en teen blootstelling aan lig beskerm word. Vars aangemaakte oplossings van *Fungizone* kan 24 uur lank teen kamer-

temperatuur bewaar word met minimum-verlies van krag. Oplossing moet egter teen blootstelling aan lig beskerm word, en na verstryking van 24 uur moet enige deel van die oplossing wat nie gebruik is nie, weggegooi word.

Indikasies: *Fungizone* vir infusie is spesifiek bedoel vir die behandeling van verspreide mikotiese infeksies, insluitende coccidioidomikose; kriptococcosis (torulose); verspreide moniliase; histoplasmose, en Noord-Amerikaanse blastomikose.

Toediening: *Fungizone* moet as 'n stadige binneaarse infusie oor 'n tydperk van ongeveer 6 uur toegedien word. Die gewone voorsorgsmaatreëls wat op binne-aarse terapie van toepassing is, behoort in ag geneem te word.

Vir binne-aarse infusieterapie: Die aanbevole konsentrasie vir binne-aarse infusie is 0.1 mg./k.s. (1 mg./10 k.s.). Die oplossing vir infusie word voorberei deur 10 k.s. Dekstrose-inspuiting 5% U.S.P. by die droë *Fungizone*-poeier te voeg. Dit verskaf 'n aanvanklike konsentrasie van 5 mg./k.s. Skud die flessie dan goed totdat u 'n heldere oplossing het. Die aanbevole konsentrasie van 0.1 mg./k.s. (1 mg./10 k.s.) word verkry deur verdere verdunning met Dekstrose-inspuiting U.S.P.

Dosis: Aangesien verdraagsaamheid vir sover dit *Fungizone* betref, van die een persoon tot die ander verskil, moet die dosis by die spesifieke behoeftes van iedere pasiënt aangepas word. Terapie word ingestel met 'n daaglikse dosis van 0.25 mg. per kg. liggaamsgewig en geleidelik vermeerder totdat die optimumpeil bereik word. Oor die algemeen kan die totale daaglikse dosis tot 1.0 mg. per kg. liggaamsgewig wissel.

Binne nierdie perke behoort die dosis gehandhaaf te word op die hoogste moontlike perk wat *nie toksiese manifestasies* soos hoofpyn, mislikheid, braking of 'n styging van bloedureumstikstof of nie-proteïenstikstof te voorskyn bring nie. In die geval van pasiënte wat ernstig siek is en geen baat by 'n daaglikse dosis van 1 mg./kg. vind nie, kan die dosis versigtig oorskry en geleidelik vermeerder word tot 'n daaglikse maksimum-dosis van 1.5 mg./kg., mits *geen toksiese effekte hulle openbaar nie*. Aangesien *Fungizone* baie stadig afgeskei word, kan pasiënte wat die hoër dosisse kry al om die ander dag behandel word.

Waarskuwing: *In geen omstandighede moet die totale daaglikse dosis* 1.5 *mg./kg. oorskry nie.*

Nadat verbetering waargeneem is, kan die daaglikse toediening van die antibioticum plek maak vir terapie al om die ander dag. Die duur van die terapie hang af van die aard en die erns van die infeksie. Kliniese ondervinding tot dusver het aangedui dat betekenisvolle verbetering in die meeste gevalle na 4 tot 8 weke se behandeling verwag kan word. Terapie oor 'n korter tydperk het skynbaar 'n minder gunstige reaksie; gevolglik is die moontlikheid van 'n nuwe aanval nie uitgesluit nie.

Voorsorgsmaatreëls: Verbygaande anoreksie, verkoue en koors word dikwels teëgekom tydens die eerste paar dae van *Fungizone*-terapie. Daarna het hierdie reaksies egter 'n neiging om af te neem en minder lastig te word. Die toediening van koorswerende en/of antihistamienmiddels is nuttig, en help om die genoemde newe-effekte te bestry. Indien koorsige reaksies tydens infusie waargeneem word, behoort die toediening van die middel gestaak te word om die pasiënt 'n kans te gee om van die episode te herstel. Hoofpyn, mislikheid en braking is vroeë toksiese bewyse dat die totale daaglikse dosis van die antibioticum verminder behoort te word. Bloedureumstikstof- (BUS) en nie-proteïenstikstof- (NPS) peile behoort gereeld tydens terapie met *Fungizone* nagegaan te word. BUS- en NPS-peile moet nie 20 mg. % en 40 mg. % onderskeidelik oorskry nie. Indien verhoogde BUS- en NPS-peile waargeneem word, moet *Fungizone*-terapie 7 dae lank of selfs langer gestaak word totdat die genoemde peile na die normale perke terugkeer. As die behandeling dan hervat word, moet dit op die laagste dosispeil geskied, d.w.s. 0.25 mg. per kg. liggaamsgewig, en dan geleidelik vermeerder word tot die optimumpeil soos onder , Dosis' uiteengesit.

Net soos met ander middels wat binne-aars toegedien word, is dit moontlik dat plaaslike ontstekingsreaksies by die inspuitingsplek of tromboflebitis by sommige pasiënte kan voorkom. Tromboflebitis kan teëgewerk word deur die konsentrasie van die infusie-oplossing tot benede 0.1 mg./k.s. te verminder, deur die infusie stadiger te laat geskied, en deur 'n dunner naald te gebruik.

Waarskuwing: *Indien ander toksiese manifestasies hul verskyning maak tydens behandeling met* Fungizone *moet terapie met hierdie middel onmiddellik gestaak word.*

'n Klein voorraad *Fungizone* is tans vir noodgevalle in Suid-Afrika beskikbaar. Voorrade is verkrygbaar van Protea Pharmaceuticals Limited, Posbus 7793, Johannesburg.

KENACORT

Squibb stel tot beskikking van die mediese professie in Suid-Afrika *Kenacort*, 'n nuwe kortikosteroïed met verhoogde terapeutiese bedrywigheid en aansienlik minder onwenslike newe-effekte.

Kenacort is Squibb-triamsinoloon. Chemies is dit die 9-alfa-fluoro-16-alfa-hidroksi-derivaat van prednisoloon. 'n Wysiging van die basiese kortikoïedstruktuur, soos in *Kenacort* bewerkstellig, het 'n versterking van die onstekingbestrydings-, hormoon- en metaboliese eienskappe tot gevolg, gepaard met 'n gelyktydige verbanning van sekere ongunstige effekte soos natriumretensie en edeem.

Vir mondelinge toediening is *Kenacort* beskikbaar in tablette van 1 en 4 mg. Albei tipes het kepies om maklike dosering in die hand te werk.

Gebruiksaanwysings: Die spesifieke eienskappe van *Kenacort* wat dit van ander glukokortikoïede onderskei, is belangrike faktore in die kliniese gebruik van hierdie samestelling. Aangesien *Kenacort* in die gewone dosisse natriumafskeiding in die hand werk en geen edeem veroorsaak nie, is die klassieke tekens van te groot dosisse kortisoon, soos 'n vermeerdering van gewig en kliniese edeem, geen aanduiding dat te groot dosisse *Kenacort* gebruik word nie. Hierdie spesifieke kenmerke van die middel maak 'n dieet met 'n klein natriuminhoud ook onnodig tydens *Kenacort*-terapie. Gedurende die eerste paar dae van *Kenacort*-toediening word die gewigsverlies en vermeerderde urinering wat met natrium-diurese geassosieer is, dikwels deur die geneesheer opgemerk, veral by pasiënte wat ly aan natriumretensie en edeem ten gevolge van vroeëre behandeling met ander glukokortikoïede, of weens 'n ander onderliggende siekte.

Kaliumaanvulling tydens *Kenacort*-toediening is nie nodig nie, aangesien die middel in die reël geen effek op die kaliumewewig het nie. Hoë bloeddruk, 'n vermeerdering van gewig, edeem en tekens van 'n kaliumtekort moet nie beskou word as 'n aanduiding dat te groot dosisse *Kenacort* toegedien word nie. Die ander voorsorgsmaatreëls en kontra-indikasies vir die gebruik van glukokortikoïed-terapie is egter ook van toepassing op triamsinoloon.

Indikasies: *Kenacort* word aanbeveel vir die behandeling van 'n groot verskeidenheid van kliniese kwale, onder meer chroniese gewrigsontsteking, brongiale asma, vasmotoriese neusslymvliesontsteking, angioneurotiese edeem, dermatoses, die leukemies, limfosarkoma, Hodgkin se siekte, verspreide lupus erythematosus, die nefrotiese sindroom, pulmonale emfiseem en pulmonale fibrose. *Kenacort* kan ook van waarde wees by die behandeling van akute slymbeursontsteking, rumatiekkoors en sekere bloeddiskrasies.

Toediening en Dosis: Optimum-dosispeile vir *Kenacort* wissel van pasiënt tot pasiënt en moet afsonderlik vir iedere pasiënt en vir die siekte onder behandeling vasgestel word. Die aanbevole aanvanklike dosis vir mondelinge terapie is 8 tot 20 mg. per dag, in verdeelde dosisse. Nadat 'n bevredigende reaksie verkry is, moet die dosis geleidelik verminder word (2 mg. al om die 2 tot 3 dae) totdat 'n optimum instandhoudingspeil bereik is.

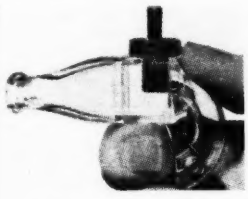
Nadere Besonderhede is Verkrygbaar van: Squibb Laboratories (Pty.) Limited, Posbus 9975, Johannesburg.

NORISODRINE-SULFAAT

VIR DIE VINNIGE VERLIGTING VAN ASMA

In *Norisodrine*-sulfaatpoeier (Abbott) is 90% van die deeltjies kleiner as 10 mikron.

Die pasiënt asem een of twee maal in uit 'n *Aerohaler* wat met *Norisodrine* gevul is; deur suiging word die *Norisodrine*-deeltjies dan in die asemhalingskanaal ingetrek.



Omdat die deeltjies van optimumgrootte is, word hulle tot in die dieptes van die asemhalingsweë getrek en kom in intieme aanraking met die slymvlies.

Broncho-krampe word byna onmiddellik gestuit.

Met behoorlike aanpassing van die dosis is daar selde enige gevaar van newe-effekte.

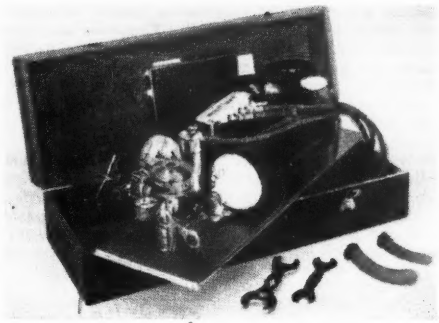
Norisodrine word veral aangedui vir die behandeling van pasiënte wat weerstand teen epinefrien bied, en diegene aan wie middels soos teofillien en die jodiede geen verligting besorg nie.

Alleenfabrikante in Suid-Afrika: Abbott Laboratories (S.A.) (Pty.) Ltd., Posbus 1616, Johannesburg.

BEWUSSYNHERSTELLING: DIE STEPHENSON MINUTEMAN

African Oxygen Limited het 'n draagbare, volkome outomatiese suurstofbewussynhersteller van 'n moderne tipe bekend as die Stephenson Minuteman beskikbaar gestel, en verskaf die volgende inligting:

Hierdie nuwe eenheid word gekenmerk deur 'n aantal merkwaardige verbeterings in vergelyking met vorige eenhede van 'n dergelike tipe:



1. 1–3 pasiënte kan gelyktydig behandel word.
2. Verstelbare asemhalingsdruk en -frekwensie vir volwassenes en kinders.
3. Suurstofbewussynherstelling of -inaseming kan verander word van 100% O_2 tot 50% O_2.
4. Geskik vir gebruik as 'n maklik vervoerbare veldeenheid of as 'n vaste muureenheid in ambulanskamers, ens. of as 'n hospitaaleenheid.
5. Voorsiening word gemaak vir twee 72-gelling suurstofsilinders van ligte staal vir veldgebruik (een blywend, en die ander 'n reserwe), sowel as vir verbinding met enige groot suurstofsilinder vir vaste of hospitaalgebruik.

'n Praktiese demonstrasie kan gereël word met enige tak van die firma African Oxygen Limited, Posbus 5404, Johannesburg, by wie nadere inligting verkrygbaar is.

BESKIKBAARSTELLING VAN DISTAQUAINE V–K

British Drug Houses kondig met genoeë die beskikbaarstelling van *Distaquaine V–K*-tablette aan. Hulle is saamgestel uit die kaliumsout van penisillien V, en word aanvanklik slegs in 'n enkele sterkte bemark —iedere tablet bevattende die gelyke van 125 mg. penisillien V-vrysuur.

Daar word allerweë aangeneem dat die oplosbaarheid van 'n mondelinge penisillienpreparaat die sleutel bied tot die absorpsietempo van daardie preparaat, en gevolglik ook tot die bloedpeile wat dit kan bewerkstellig. Aangesien die kaliumsout van fenoksimetielpenisillien (*Distaquaine V–K*) 'n produk is wat makliker as die oorspronklike *Distaquaine V*-preparate oplos, het kliniese ondervinding aangetoon dat bloedpeilhoogtepunte en hoër konsentrasies gouer deur die vinniger absorpie daarvan teweeggebring kan word as die pasiënt dit voor 'n maaltyd neem.

Hoewel daar geen bewys is nie dat *Distaquaine V–K* uit 'n terapeutiese oogpunt aansienlik doeltreffender is as die vrysuur-produkte van die *Distaquaine V*-reeks wat betroubare en standhoudende resultate oplewer of hulle nou al vóór of ná 'n maaltyd aan 'n pasiënt gegee word, bestaan daar tog 'n behoefte aan 'n produk wat in staat is on hoë bloedpeile vinniger tot stand te bring. Hoe dit ook al sy, die geneesheer sal voortaan kan kies tussen *Distaquaine V–K*-tablette en *Distaquaine V*-preparate, na gelang van die behoeftes van sy pasiënt en die stadium wat bereik is deur die infeksie wat behandel moet word.

Distaquaine V–K kan nou voorgeskryf word uit die volgende verpakkings:

Distaquaine V–K-tablette, 125 mg.; kartondosies van 12; bottels van 100; bottels van 500.

PREPARATIONS AND APPLIANCES

FUNGIZONE FOR INFUSION

Fungizone *is a potent antibiotic. The physician planning to use this drug should be fully acquainted with its properties.*

Fungizone is Squibb Amphotericin B, a new antifungal antibiotic. For intravenous use, *Fungizone* is supplied as a sterile lyophilized powder in vials providing 50 mg. amphotericin B and a total of approximately 46 mg. sodium desoxycholate with sodium phosphates as a buffer. For administration by intravenous infusion, the dry *Fungizone* powder should be dissolved in Dextrose Injection 5% U.S.P.

Amphotericin B is derived from an unidentified *Streptomyces* species found in a South American soil sample. The isolation, crystallization and biochemical characterization of the antibiotic was achieved in the laboratories of the Squibb Institute for Medical Research. Since crystalline amphotericin B is practically insoluble in aqueous media, an amphotericin B sodium desoxycholate mixture was developed resulting in a solubilized material. Clinical evidence indicates that *Fungizone* is effective against a wider variety of deep-seated fungi and yeasts than any other antifungal agent now available for use in man. The antibiotic is without effect on bacteria. Demonstrable blood levels of the antibiotic persist for 18 hours after intravenous infusion is discontinued, suggesting that the antibiotic is slowly excreted by the kidneys.

Storage: Vials of dry *Fungizone* powder should be stored in the refrigerator, protected against exposure to light. Freshly prepared solutions of *Fungizone* may be stored at room temperature for 24 hours with a minimum loss in potency; solutions must be protected against exposure to light and any unused material should be discarded after 24 hours.

Indications: *Fungizone* for infusion is specifically intended for the treatment of disseminated mycotic infections including coccidioidomycosis; cryptococcosis (torulosis); disseminated moniliasis; histoplasmosis; and North American blastomycosis.

Administration: *Fungizone* should be administered by *slow* intravenous infusion over a period of approximately six hours, observing the usual precautions for intravenous therapy.

For intravenous infusion therapy: The recommended concentration for intravenous infusion is 0.1 mg./c.c. (1 mg./10 c.c.). The infusion solution is prepared by adding 10 c.c. Dextrose Injection 5% U.S.P. to the dry *Fungizone* powder to provide an initial concentration of 5 mg./c.c.; shake the vial vigorously until a clear solution is obtained. The recommended concentration of 0.1 mg./c.c. (1 mg./10 c.c.) is then obtained by further dilution with Dextrose Injection U.S.P.

Dosage: Since tolerance to *Fungizone* varies individually, dosage must be adjusted to the specific requirements of each patient. Therapy should be instituted with a daily dose of 0.25 mg./Kg. of body weight and *gradually increased* until an optimum level is attained. Generally, total daily dosage may range up to a level of 1.0 mg./Kg. of body weight.

Within this range, dosage should be maintained at the highest possible level that is *not accompanied by toxic manifestations* (headache, nausea, vomiting, or rise in blood urea nitrogen or non-protein nitrogen). In seriously ill patients not benefited by daily doses of 1 mg./Kg. this dose may be exceeded cautiously and gradually up to a maximum daily dose of 1.5 mg./Kg., *provided no toxic effects are encountered.* Since *Fungizone* is excreted very slowly, therapy may be given on alternate days in patients on the higher dosage schedule.

Caution: *In no circumstances should total daily dosage of 1.5 mg./Kg. be exceeded.*

When improvement is observed, daily administration of the antibiotic may be substituted with therapy on alternate days. Duration of therapy depends on the nature and severity of the infection. In clinical experience to date, significant improvement has been observed in most instances after 4 to 8 weeks of treatment. A shorter period of therapy appears to produce a less favourable response and may lead to relapse.

Precautions: Transient anorexia, chills and fever are frequently encountered during the first few days of *Fungizone* therapy; thereafter, however, these reactions generally diminish and tend to be less troublesome. Administration of antipyretics and/or antihistamines is helpful in minimizing these side effects. If febrile reactions occur during the infusion, administration of the drug should be interrupted to allow the patient to recover from the episode. Headache, nausea and vomiting are early toxic manifestations of the antibiotic requiring a reduction in the total daily dosage. Blood urea nitrogen (BUN) and non-protein nitrogen (NPN) levels should be checked routinely during *Fungizone* therapy. BUN and NPN levels should not exceed 20 mg. % and 40 mg. % respectively. If increased BUN or NPN levels are observed, *Fungizone* therapy should be interrupted for 7 days or longer until the level returns to normal limits. When medication is reinstated, therapy should be resumed by starting with the lowest dosage level, i.e. 0.25 mg./Kg. of body weight and increasing gradually to an optimum level as outlined under 'Dosage'.

As with other intravenously administered drugs, local inflammatory reactions at the injection site or thrombophlebitis may occur in some patients.

Thrombophlebitis may be minimized by decreasing the concentration of the infusion solution below 0.1 mg./c.c., reducing the rate of infusion and using a smaller gauge needle.

Caution: *If other toxic manifestations occur during the course of therapy, discontinue* Fungizone *immediately.*

A small supply of *Fungizone* is now available in South Africa for emergency cases. Stocks are kept at Protea Pharmaceuticals Limited, P.O. Box 7793, Johannesburg.

KENACORT

Squibb introduce *Kenacort* to the medical profession in South Africa—a new corticosteroid which offers enhanced therapeutic activity and significant reduction of undesirable side-effects.

Kenacort is Squibb Triamcinolone. Chemically, it is the 9-alpha-fluoro-16-alpha-hydroxy derivative of prednisolone. Modification of the basic corticoid structure as achieved in *Kenacort* has resulted in a potentiation of the anti-inflammatory, hormonal and metabolic properties with a concomitant abrogation of certain unwanted effects such as sodium retention and edema.

For oral administration, *Kenacort* is available in tablets of 1 and 4 mg. Both potencies are scored to provide flexibility of dosage.

Rationale for Use: The specific properties of *Kenacort* which differentiate it from other glucocorticoids are important factors in the clinical utility of this compound. Since *Kenacort*, in the usual doses, promotes sodium excretion and does not cause edema, the classic signs of cortisone overdosage such as increase in weight and clinical edema, cannot be employed as criteria for *Kenacort* overdosage. These specific attributes of the drug also obviate the need for a low sodium diet during *Kenacort* therapy. During the first few days of *Kenacort* administration, weight loss and increased urinary output associated with sodium diuresis occur frequently, particularly in patients who have sodium retention and edema as a result of previous treatment with other glucocorticoids or due to an underlying disease. Potassium supplementation during *Kenacort* administration is not required since the drug generally does not affect potassium balance. Hypertension, increase in weight, edema, and signs of potassium deficiency should not be used as an indication of overdosage when *Kenacort* is used. However, other precautions and contraindications for glucocorticoid therapy also apply with triamcinolone.

Indications: *Kenacort* is recommended for the management of a wide variety of clinical disorders. These include rheumatoid arthritis, bronchial asthma, vasomotor rhinitis, angioneurotic edema, dermatoses, the leukemias, lymphosarcoma, Hodgkin's disease, disseminated lupus erythematosus, the nephrotic syndrome, pulmonary emphysema and pulmonary fibrosis. *Kenacort* may also be of value in the treatment of acute bursitis, rheumatic fever and in certain blood dyscrasias.

Administration and Dosage: Optimum dosage levels for *Kenacort* vary from patient to patient and must be determined individually for each patient and for the disease under treatment. The suggested starting dose for oral therapy is 8 to 20 mg. per day, in divided doses. When a satisfactory response is obtained, dosage should be reduced gradually (2 mg. every 2 to 3 days) until an optimum maintenance level is achieved.

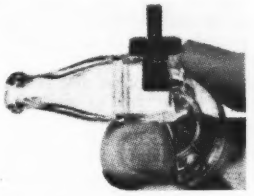
Further Particulars are Available from: Squibb Laboratories (Pty.) Limited, P.O. Box 9975, Johannesburg.

NORISODRINE SULPHATE

FOR RAPID RELIEF OF ASTHMA

In *Norisodrine* Sulphate Powder (Abbott), 90% of the particles are under 10 microns in size.

The patient inhales once or twice from an *Aerohaler* charged with *Norisodrine;* suction then draws the *Norisodrine* particles into the respiratory tract.



Because the particles are of the optimal size, they are drawn into the depths of the respiratory passages and come into intimate contact with the mucous membrane. Bronchospasm is aborted almost immediately.

With proper adjustment of dosage there is seldom any risk of side effects.

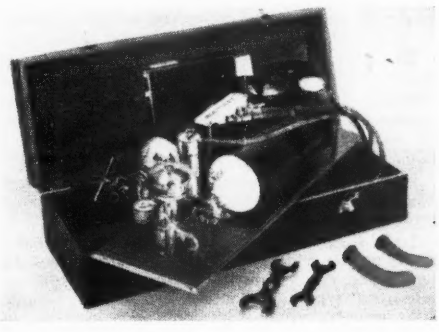
Norisodrine is particularly indicated for patients who are epinephrine-fast, and for those who do not obtain relief with such drugs as theophylline and iodides.

Sole South African Manufacturers: Abbott Laboratories (S.A.) (Pty.) Ltd., P.O. Box 1616, Johannesburg.

RESUSCITATION: THE STEPHENSON MINUTEMAN

African Oxygen Limited have introduced an up-to-date type of a portable fully automatic oxygen resuscitator under the name the Stephenson Minuteman and supply the following information:

This new unit incorporates a number of remarkable improvements over previous units of a similar kind:



1. Treatment of 1–3 patients simultaneously.

2. Adjustable breathing pressure and frequency for adults and children.

3. Oxygen resuscitation or inhalation variable, from 100% O_2 to 50% O_2.

4. Suitable as a highly portable field unit or as a stationary wall unit in ambulance rooms, etc. or as a hospital unit.

5. Allows for two 72-gallon light-weight steel oxygen cylinders for field use (one permanent, one spare) and the connecting of any large-size oxygen cylinder for stationary or hospital use.

A practical demonstration can be arranged by any branch of Messrs. African Oxygen Limited, P.O. Box 5404, Johannesburg, from whom further information may be obtained.

INTRODUCING DISTAQUAINE V–K

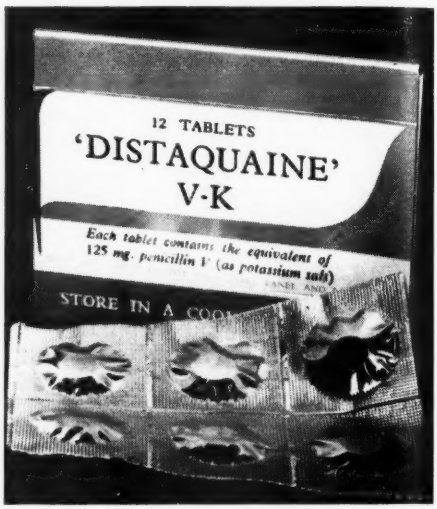
British Drug Houses have pleasure in announcing the availability of *Distaquaine V–K* tablets. These consist of the potassium salt of penicillin V and are initially being marketed in one strength only—each tablet containing the equivalent of 125 mg. of penicillin V free acid.

It is accepted that the solubility of an oral penicillin preparation is the key to the rate of absorption and thus to blood levels achieved. Since the potassium salt of phenoxymethylpenicillin (*Distaquaine V–K*) is a product of greater solubility than the original *Distaquaine V* preparations, clinical experience has shown that its faster absorption will produce earlier peak blood levels and higher therapeutic concentrations if administered to a patient before food.

Although there is no evidence of a substantial increase in therapeutic efficiency over the free acid products (*Distaquaine V* series), which have given reliable and consistent results whether administered to patients before or after food, the demand for a product to produce earlier peak blood levels does exist. However, both *Distaquaine V–K* tablets and the *Distaquaine V* preparations will now be available to give a choice of product, dependent upon the need of the patient and the stage of infection to be treated.

Distaquaine V–K may now be prescribed from the following packs:

Distaquaine V–K Tablets 125 mg.: Cartons of 12; Bottles of 100; Bottles of 500.



CORRESPONDENCE

POLIOMYELITIS AND EPIDEMIOLOGY

To the Editor: I was rather startled to read the following sentence in Dr. Braudo's article on poliomyelitis:[1] 'Since poliomyelitis occurs in epidemics, it must be an infectious disease.' Epidemics of scurvy occurred on board ship in Van Riebeeck's day. War, concentration camps, etc. are liable to result in epidemics of pellagra. Contamination of food with triorthocresyl phosphate is likely to result in an epidemic of lower motor neurone paralysis.[2]

Dr. Braudo's interesting article suggests to me that a controlled investigation would be provided by:

i. The '6 instances of multiple infection with various form of the disease,' in one household from his series, and the '30 instances of multiple infection,' from the 1947–8 Johannesburg epidemic;

ii. A comparable number of families or households in which only one person suffered from poliomyelitis during the same epidemic.

It is suggested that a sanitary inspector and a social worker visit such homes to investigate, e.g. family history of poliomyelitis, possible avenues of infection, type of housing, crowding, sleeping arrangements, feeding habits, recreational habits. Investigation by a medical practitioner of the nutritional status of the families (patient as well as contacts) might also prove to be profitable. The results might well throw light on the problem (indicated by Sabin and quoted by Dr. Braudo) whether poverty and poor hygiene are important factors. Two questions remain to be answered:

i. What conditions in some families promote a higher rate of infection?

ii. What conditions in some families promote a higher rate of illness following infection?

REFERENCES

1. Braudo, J. L. (1958): This Journal, **4**, 169.
2. Sampson, B. R. (1942): S. Afr. Med. J., **16**. 1.

B. J. Kaplan,
Senior Lecturer in Social Medicine.

Department of Medicine,
Medical School,
Mowbray, C.P.